THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI -600032.

FACTORS PREDICTING OUTCOMES AFTER SURGERY IN POST MENINGITIC HYDROCEPHALUS IN CHILDREN UNDER 12 YEARS

Dissertation submitted in partial fulfillment

of the requirements of

M.Ch BRANCH II NEUROSURGERY (3 YEARS)

EXAMINATIONS – AUGUST 2013

INSTITUTE OF NEUROLOGY

MADRAS MEDICAL COLLEGE & RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL

CHENNAI-600003.

AUGUST -2013

CERTIFICATE

This is to certify that this dissertation entitled **"FACTORS PREDICTING OUTCOMES AFTER SURGERY IN POST MENINGITIC HYDROCEPHALUS IN CHILDREN UNDER 12 YEARS"** submitted by **Dr.K.Balasubramani** appearing for **M.Ch (Neurosurgery)** degree examination in August 2013 is a original bonafide record of work done from August 2010 to March 2013 by him under my guidance and supervision in partial fulfillment of requirement of the Tamil Nadu Dr.M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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DECLARATION

I, Dr. K.Balasubramani, solemnly declare that this dissertation **"FACTORS PREDICTING OUTCOMES AFTER SURGERY IN POST MENINGITIC HYDROCEPHALUS IN CHILDREN UNDER 12 YEARS"** was done by me at the department of neurosurgery at Institute of Neurology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai under the guidance and supervision of the Professor of Neurosurgery, Institute of Neurology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-3, between 2010 and 2013.

This dissertation is submitted to the Tamil Nadu Dr.M.G.R. Medical University, Chennai-600032 in partial fulfilment of the University requirements for the award of the degree of M.Ch., Neurosurgery.

Place : Chennai

Date : 26-03-13

(Dr.K.Balasubramani)

ACKNOWLEDGEMENT

I owe my thanks to **THE DEAN**, Madras Medical College, Chennai, for permitting me to utilize the facilities and conducting this study and the members of Ethical Committee for their role.

I am extremely grateful to **Prof. K.DEIVEEGAN, M.S., M.Ch.,** Professor of Neurosurgery and Head of the Department, Institute of Neurology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai, for his constant encouragement and guidance throughout the study and periodic reviews.

I sincerely thank all the Professors of our department **Prof.V.SUNDAR, Prof.V.G.RAMESH, Prof.S.SUNDARAM, Prof.K.MAHESHWAR, Prof.S.D.SUBBIAH, Prof.C.SEKAR, Prof.S.SYAMALA, Prof.J.V.MAHENDRAN, Prof.RANGANATHAN JOTHI, Prof.JAGANNARAYANA** for helping me with their time and advice during this study.

I am indebted to all my assistant professors for their support, guidance and help without which it would had been difficult to carry out this study.

The support and sacrifice of my family need special mention.

The blessings of Almighty without which this work would not have been possible is acknowledged with gratitude.

TABLE OF CONTENTS

ABBREVIATIONS USED

ATT – Anti tubercular therapy

GCS –Glasgow Coma Scale

TBM – Tuberculous Meningitis

CSF – Cerebrospinal fluid

AFB – Acid Fast Bacilli

C/S – Culture and Sensitivity

INTRODUCTION

Post meningitic hydrocephalus is a common and perplexing disease of the developing world. It is almost always seen in patients with meningitis for 4 weeks[1]. Meningitis maybe pyogenic, tubercular (which is very common), viral and rarely fungal or parasitic.

The most definitive treatment for hydrocephalus, is surgical however active or severe the meningitic process maybe. Surgical options are various forms of shunting into the myriad body cavities, of which ventriculo peritoneal shunt is the most common, feasible and proven surgery.

The high incidence of post meningitic hydrocephalus, their varied presentations, the different specialities handling these cases before referral, and its sequelae necessitates a study on prognostication which will help in triage, prompt management and referral and the counselling of the relatives. Hence a study is conducted in an attempt to analyse the factors predicting outcome after surgery in post meningitic hydrocephalus.

AIMS AND OBJECTIVES

1. To categorize the various factors influencing the outcome after surgery in postmeningitic hydrocephalus
2. To analyze these factors and to find out the statistical significance in affecting the outcome
3. To determine if any single factor significantly predicts the outcome of the surgery
4. To enable the neurologists and neurosurgeons to understand and correlate the varied clinical manifestations.

REVIEW OF LITERATURE

Hydrocephalus is an abnormal expansion of cavities (ventricles) within the brain that is caused by the accumulation of cerebrospinal fluid. Hydrocephalus comes from two Greek words: hydros means water and cephalus means head[2]. Hydrocephalus maybe due to either an increased production of CSF in the choroid plexus of the ventricles or a decrease in the rate of absorption of CSF from the system.

Hydrocephalus maybe classified functionally as

- ***Communicating hydrocephalus***, wherein the CSF circulation is preserved at the ventricular level and the block is at the level of arachnoid granulations
- ***Non communicating hydrocephalus***, where the CSF pathway is obstructed before the arachnoid granulations which can be at the level of aqueduct of sylvius (commonest), foramina of Luscka, formaen of Magendie, foramen of Monro (causing unilateral enlargement) or cistern level.

It can also be classified as congenital or acquired. Of the various causes of acquired hydrocephalus post infectious is a very common etiology[3], and more so in our population. Among the post infectious hydrocephalus tuberculous meningitis is the commonest cause in the developing world[4], especially in our country.

Pathology of post meningitic hydrocephalus

Meningitis is defined as inflammation of the membranes that surround the brain and spinal cord[5]. It is the process of inflammation of the meningeal covering of the brain, especially the leptomeninges composed of the piamater and arachnoid.

Tuberculous meningitis

Tuberculous meningitis starts as small foci of subpial or subependymal regions (Rich foci)[6]. When these foci rupture into the subarachnoid space and the basal cisterns, there is extensive inflammation of the basal cisterns and the subarachnoid space, which is the basic pathology of tuberculous meningitis.

In the early stages of the disease there is a gelatinous exudate blocking the basal cisterns namely the (prepontine cistern, the interpeduncular cistern and the ambient cistern) causing communicating hydrocephalus[7]. Later on, it proceeds to scarring and permanently impairs the CSF circulation in the brain of the+ patient. Blocking of arachnoid granulations is another contributory mechanism to the

development of communicating hydrocephalus. The exudates and scar may also obstruct the outflow to the fourth ventricle causing obstructive hydrocephalus. There is an additional contribution of inflammation of the choroid plexus causing hyper secretion of CSF, causing increased production. Thus hydrocephalus in TB meningitis can either be of the communicating or the non communicating types, of which the former is supposedly more common[8].

The basal exudates also encase the basal vessels particularly of the vertebral system, involve them and produce vasculitis which causes infarcts in the basal nuclei and the deep white matter of the brain[9]. Thus the gamut of presentations and symptomatology in tuberculous meningitis can be explained. Ultimately upto 87% of patients land up in hydrocephalus, more so in children[10].

Clinical presentation

There is no specific single diagnostic clinical entity to diagnose tuberculous meningitis with hydrocephalus. Tuberculuos meningitis is suspected when a combination of findings - usually fever > 2 weeks, altered sensorium, seizures with prodromal anorexia with or without loss of appetite and CSF findings of lymphocytosis are seen in cases of TB meningitis[11, 12, 13, 14, 15, 16, 17].

In younger children, failure to thrive, loss of weight, loss of appetite, irritability and vomiting maybe seen[18]. History of recent contact is significant. Seizures and focal deficits like cranial deficits and hemiplegia maybe seen[19].

Laboratory features

Blood parameters may show non specific changes like Elevation of ESR and lymphocytosis. CSF analysis remains the cornerstone among the laboratory parameters in diagnosis. CSF may show AFB positive Tuberculous bacteria albeit in few children due to the difficulty in obtaining adequate sample volumes.

Normal CSF volume in various ages[20], [21],[22]

	Rate of production (ml/hr)	Volume(ml)	Safe volume to tap (ml)
Adult	22	150-170	15-17
Adolescent	18	120-170	12-17
Young child	12	100-150	10-15
Infant	10	60-90	6-9
Term neonate	1	20-40	2-4

CSF is usually analysed for AFB staining and C/S, CSF biochemistry to look for glucose and protein, CSF cytology and cell count.

CSF microbiology

CSF microbiology for demonstration of AFB positive bacteria, though the diagnostic test, is seldom positive especially in children, as it is dependent on the quantity of CSF taken for the yield, as noted above the volume to be taken is substantially less in younger children. Positive yield is dependent on CSF of atleast 6ml[23].

CSF biochemistry and cytology

CSF biochemistry shows elevated protein levels 60-700 mg/dl and glucose 20-40% of the serum with cell count of 50-500/cu mm with lymphocytic predominance[24].

Imaging

Computed Tomography of the brain still remains the cornerstone imaging in cases of tuberculous meningitis due to its availability and cost effectiveness.Contrast enhanced CT of the brain is the imaging of choice. The commonest finding accompanying hydrocephalus was basal enhancement (80%) as can be understood from the pathology of tuberculous meningitis[25]. Infarctions are seen in 20% patients, which is due to vasculitis, mostly involving the basal ganglia and the territories of the medial striate and thalamoperforating arteries[26],[27],[28].Tuberculomas may also be seen which help in the diagnosis[29].

MRIs due to their cost and time consumption are usually done to pick up changes in early disease and to differentiate them from neurocysticercosis. Early

small infratentorial lesions are seen in MRI[30], [31], [32]. Large lipid lactate peak and choline/creatinine ratio >1 are used to differentiate them for neurocysticercosis[33], [34]

Diagnosis

Diagnosis is done not based on a single entity, rather, a combination of clinical features CSF and radiology

Various studies have published many diagnostic rules for diagnosis which can be summarised as follows

Rule	Age Group	Symptoms	Remarks
Children rule[35]	1 month to 12 years	• > 6 days of symptoms • Optic atrophy • Abnormal movements • Focal deficit • PMNs < half the TLC in CSF	If ➢ ≥ I variable then (98% sensitivity & 44% specificity) ➢ ≥ 2 variables then (77% sensitivity & 57% specificity) ➢ ≥ 3 variables then (55% sensitivity & 98% specificity)
Children & adult rule[36]	5 months – 56 yrs	➢ > 5 days symptoms ➢ Clear CSF ➢ Lymphoctes > 30% of TLC in CSF ➢ CSF protein > 100 mg/dl	➢ If ≥ 2 variables then (93% sensitivity & 77% specificity)

Clinical case definition of tuberculous meningitis devised by *Doerr* et al[37]

Abnormal neurological signs and symptoms with ≥ 2 of the following 1. H/O significant contact with adult with contagious TB 2. PPD >10mm or PPD >5mm with H/O close contact with infected person 3. CSF abnormalities with no evidence of other infections 4. CT consistent with TB

The diagnosis of TBM was established as per *Ahuja* et al[38] (38) criteria is as follows.

Criteria for the diagnosis of TB meningitis	The 4 sub-criteria described above have been incorporated into 4 groups in descending order of sensitivity:
A. Clinical (i) fever and headache lasting for more than 14 days (mandatory) (ii) vomiting, alteration of sensorium or focal deficit (optional) **B. Cerebrospinal fluid** (i) pleocytosis with more than 20 cells, predominantly (greater than 60%) lymphocytes (ii) protein greater than 100 mg/dL, sugar less than 60% of corresponding blood Sugars (iii) negative India ink studies and cytology for malignant cells (in relevant situations) **C. Radiological** CT studies of the head showing 2 or more of the following: (i) exudates in basal cisterns or in Sylvian fissures (ii) hydrocephalus (iii) infarcts (iv) gyral enhancement **D. Evidence of extra-neural tuberculosis** Active tuberculosis of lungs, gastrointestinal tract, urogenital tract, lymph nodes, skeletal system or skin as evidenced by appropriate radiological or microbiological tests or by the presence of caseation necrosis on histopathological examination	**1. Definite tuberculosis meningitis:** (i) clinical criteria (A); (ii) bacterial isolation from CSF or diagnosis at autopsy **2. Highly probable tuberculosis meningitis** (i) Clinical criteria (A); (ii) All 3 of (B) and (C) and (D); and good response to antituberculosis treatment **3. Probable tuberculosis meningitis** (i) Clinical criteria (A); (ii) Any 2 of B, C and D **4. Possible tuberculosis meningitis** (i) Clinical criteria (A); (ii) Any one of (B) (C) and (D)

Grading of the disease

Grading of the disease helps in prognostication and to decide on the treatment. MRC grading system[39] is as below

Stage 1	Fully conscious , no paresis
Stage 2	Decreased consciousness, localizing to pain
Stage 3	Deeply comatose

Another system to assess the patients especially with post meningitic hydrocephalus, namely the **Modified Vellore Grading System** is also proven to be more beneficial and accurate[40]

Grade 1	GCS 15, headache, vomiting, fever +/- neck stiffness & no neurological deficits
Grade 2	GCS 15 & Neurological deficits
Grade 3	GCS 9 to 14 +/- neurological deficit
Grade 4	GCS 3 to 8 +/- neurological deficit

Treatment

Treatment consists of starting anti tuberculous drug regime and surgical procedure for CSF diversion, namely ventriculoperitoneal shunt. Anti tuberculous drugs and their regime

Of the first line drugs used in TB Isoniazid has a good penetration[41],[42] into the CSF and it has good bactericidal action[43]. Though the penetration of Rifampicin is less its necessity is proven in many cases. Therefore high doses of Rifampicin are usually recommended. Pyrazinamide is also well absorbed and reaches high concentrations in the CSF. When adding the fourth drug in the regime their potential complications, namely renal toxicity for streptomycin and retrobulbar neuritis in ethambutol should be considered and catered. Various other second line drugs are also used in other parts of the world. Adjunctive corticosteroids are found to be helpful and recommended in all ATT regimes[44].

The ATT regime recommended by IAP is as follows[45] Two months of intensive phase comprising of a four drug regime followed bty 10 months of three drugs (2HRZE + 10HRE)

Drug	Daily therapy (mg/kg)	Intermittent therapy (mg/kg)
Isoniazid (H)	5+	15
Rifampicin (R)	10	15
Pyrazinamide (Z)	25	30
Ethambutol (E)	20	30
Streptomycin (S)	20	30
Prednisolone	1	
Isoniazid should never be given<5mg/kg body wt Prednisolone is given for 4-8 weeks daily and tapered.		

Pyogenic meningitis

Bacterial meningitis is the inflammation of the lepto meninges due to bacterial infection.

Etiology: Depending on age the commonest pathogens are[46] (46)

- < 2 months of age
  - Escherichia coli and other Gram negative enteric bacilli,
  - Group B Streptococcus
  - Listeria monocytogenes
- > 2 months to 12 years
  - Hemophilusinfluenzae type b
  - Streptococcus pneumoniae
  - Neisseria meningitidis.

The causative organism and its virulence may vary depending on the status of Hib vaccination and immunity of the children. Staphylococcus, Salmonella,

Pseudomonas, Enterococci etc have been reported in various ages. Many times demonstration of causative organisms maybe difficult, possibly due to the empirical antibiotics started for suspected meningitis and unavailability of required infrastructure[47], [48]

Pathogenesis

Acquisition of pathogens maybe either from[5] infected genital secretions – in neonates, Colonization of upper respiratory tract- infants and older children or direct inoculation of bacteria resulting from trauma, skull defects with CSF leaks, congenital

dura defects such as a dermal sinuses or meningomyelocele, or extension from a suppurative parameningeal focus.

Whichever route the bacterial pathogens maybe acquired, there is a bacteremia followed by penetration of the blood brain barrier resulting in the inoculation of the organisms in the subarachnoid space. The mechanism penetration varies among different organisms which include specialised adhesive proteins, to facilitated transport using the natural mechanisms and transcellular transport of the organisms[49].

There is an intense inflammation produced by lipopolysaccharides and peptidoglycan secreted by gram negative and gram positive bacteria respectively. This in turn starts a turn of eventual cascade which ultimately produces neuronal death, cerebral edema and hydrocephalus leading to death if left uninterrupted[50], [51], [52], [53], [54]

Clinical features

The presentation varies depending on the age. Unlike adults the the classical triad of fever headache and nuchal rigidity are seldom observed in children[55], [56]. Usually in neonates and infants, the symptoms are ill defined and maybe any of the following like fever, irritability, seizures, paradoxical irritability, vomiting, high pitched cry and lethargy associated with tense fontanelles[46].

Seizures are more common with H.influenza b and Streptococcus pneumonia[57]. Petechial rashes and purpurae are commonly seen in meningococcal meningitis.

Older children may complain of headache and blurring of vision, and other features of increased ICP and meningeal irritation.

Laboratory findings

CSF analysis remains a main stay of investigation in pyogenic meningitis.

CSF in pyogenic meningitis is usually sent for biochemical analysis, cytology and microbiology

CSF biochemistry

CSF glucose is usually more profoundly reduced in pyogenic meningitis than TB meningitis. CSF serum glucose ratio is reduce to less than 60% in neonates and less than 40% in children > 2 months of age[58], [59]

CSF cell count & cytology

Cell count is elevated and in the order of upto 20000 cells /cu mm, with a predominance of neutrophils[24]

CSF microbiology

Demonstration of bacteria by gram stain and Culture sensitivity remains the gold standard in pyogenic meningitis though most often CSF becomes sterile except in case of gram negative organisms after the first of antibiotics which is quiet often seen[60].

Diagnosis

Diagnosis of bacterial meningitis is done by considering clinical features and correlating them with laboratory findings. A combination of systemic signs of acute onset, with CSF of high opening pressure and very high cells with a profound decrease in glucose goes more towards pyogenic meningitis. CSF microbiological evidence confirms the diagnosis

Treatment

Treatment starts with an empirical course of antibiotics which are modified according the organisms isolated. The regime[61,69] recommended is

- Neonates < 3 weeks – ampicillin + aminoglycoside or cefotaxime
- Neonates > 3 weeks – vancomycin or nafcillin +cefotaxime or ceftriaxone +/- aminoglycosides
- Older children – ceftriaxone or cefotaxime +/- vancomycin

After getting the results of culture sensitivity antibiotics depending on the isolate are given.

Surgery

Before the advent of reliable shunt systems various methods were used to treat like repeated tapping through burrhole, suboccipital cranniectomy, open ventriculostomies and subarachnoid shunts[62]. But later on ventriculoatrial[63], and by 1980s ventriculoperitoneal shunting became the procedure of choice of CSF diversion after the advent of reliable shunt systems. Early shunt surgery is advocated[40], [64] and found to be beneficial in most of the patients. Some centres have shown Endoscopic Third Ventriculostomy to be useful in selected cases[65], [66], [67].

Other causes of meningitis

Post infectious hydrocephalus has been found to be seen in other rare causes i.e. fungal meningitis, viral and parasitic meningitis.

Fungal meningitis - aseptate fungi, namely absidia, mucor and rhizopus which are usually seen in diabetic and immunocompromised individuals produce an aggressive necrotizing vasculitis of the sinus extending into the cranial cavity and the brain. Hydrocephalus is seen very rarely and usually the treatment is aggressive local resection followed by amphotericin which is the drug of choice, shunting procedures usually have no role in the treatment. Aspergillus usually produces a space occupying lesion and it is treated by antifungal agents and surgery if required.

Viral meningitis – viral infections predominantly produce an encephalitis rather than a meningitis and they seldom produce hydrocephalus.

Parasitic – neurocysticercosis is a common infection in this part of the world, but they produce multiple small enhancing lesions and meningitis is rarely reported due to the destruction of the cysts which induce an antigenic response. Hydrocephalus is usually due to an intraventricular cyst obstruction and post meningitic hydrocephalus due to neurocysticercosis are rarely reported.

MATERIALS AND METHODS

This is a study of 65 patients, who had taken treatment for meningoencaphalitis with hydrocephalus admitted at the department of neurosurgery, Madras Institute of Neurology, Madras Medical College, Rajiv Gandhi Government General Hospital from December 2010 to December 2012.

The following patients were **included** in this study

1. Patients below 12 years of age
2. Patients admitted with meningitis and hydrocephalus
3. Patients admitted with history of meningitis and hydrocephalus
4. Patients who had undergone ventriculoperitoneal shunt surgery for hydrocephalus
5. Children who were neurologically and developmentally normal till the disease
6. Children with no other co morbidities

The following patients were **excluded** from the study

1. All other causes of hydrocephalus
2. Patients who did not undergo shunt surgery
3. Patients more than 12 yrs of age
4. Patients who could not be followed up for atleast 3 months

Using standardised data collection methods all the patients admitted with post meningitic hydrocephalus in the paediatric neurosurgery ward were analysed in this study. Subsequently the imaging and their reports were collected. Follow up data for the next three months were also collected. A total of 65 cases included. The data of all these patients were taken into the study. The factors taken into observation were grouped as follows

1. Clinical factors
2. Laboratory factors

3. Imaging
4. Grading
5. Time interval between admission and surgery

Clinical factors: Factors observed were

1. Age of the patient
2. Sex of the patient
3. Duration of illness & presence of fever
4. Presence of seizures
5. Consciousness level on admission using GCS on admission
6. Focal neurological deficits
7. Modified vellore grading of the patient

Age of the patient

Patients are categorised as follows based on age

- Neonates < 1 month
- Infants upto 1 year
- >1 year of age

Sex of the patient

Sex distribution was taken into account to observe and analyse the incidence and outcomes

Duration of illness

Duration of illness was recorded based on the history given by the patient on admission and the in-patient records. Duration of illness was correlated with outcome of the patient

Seizures

Seizures both generalised and focal and their variations were taken into account.

Consciousness level on admission

The consciousness level of the patients were recorded on the basis of the Glasgow coma scale appropriate for their age groups, as discussed below

Children's coma scale <4 yrs of age[68]

Points	Best eye	Best verbal		Best motor
6	-	-		Obeys
5	-	Smiles, oriented to sound, follows objects, interacts		Localizes pain
4	Spontaneous	***Crying***	***interaction***	Withdraws to pain
		Consolable	Inappropriate	
3	To speech	Inconsistently consolable	Moaning	Decorticate
2	To pain	Inconsolable	Restless	Decerebrate
1	None	None	None	None

The GCS on admission was recorded and the patients were grouped as below

- GCS 15
- GCS 9 to 14
- GCS ≤ 8

This grouping of patient was done to segregate patients who were fully conscious in one end and comatose patients on the other end with all other patients kept as intermediate and in taking to account the vellore grading of hydrocephalus[40]

Focal neurological deficits

Presence of focal neurological deficits were noted and their relationship with outcome was analysed. Focal neurological deficits were arbitrarily grouped as follows – cranial nerve palsy, hemiparesis /focal weakness or hemiplegia / multiple neurological deficits [12].

Laboratory factors

CSF parameters were taken into account which were

1. CSF biochemistry
2. CSF cytology and cell count
3. CSF microbiology

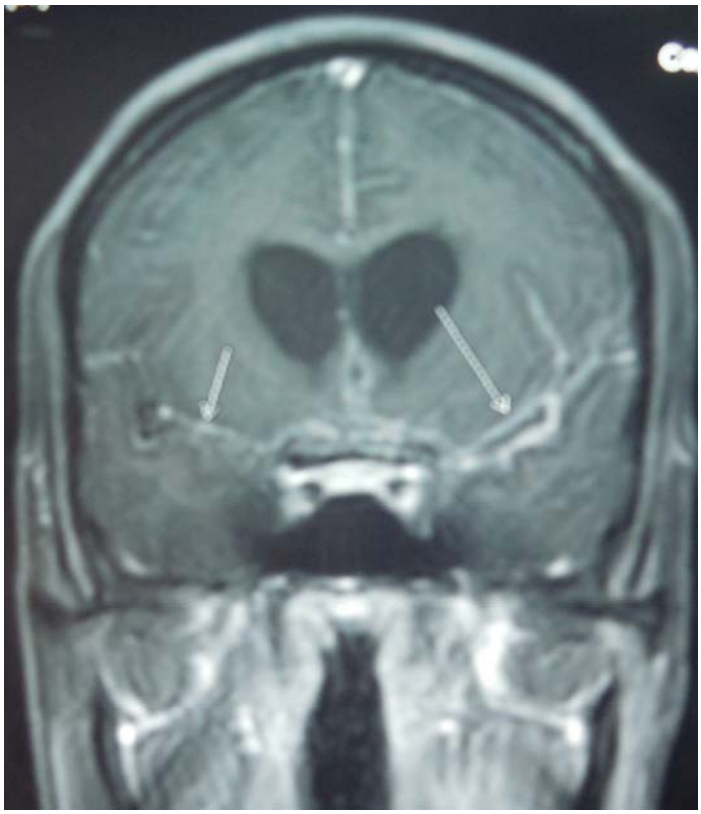


FIGURE NO: 1

CSF biochemistry

CSF glucose and protein values were noted. CSF glucose levels were compared with serum glucose levels to know the percentage of CSF glucose reduction.

CSF protein values were noted and diagnosis was confirmed and the protein values in these patients were grouped into two

CSF protein > 200 mg/dl

CSF protein ≤ 200 mg/dl

The two groups were compared with outcome and analysed.

CSF microbiology

CSF was being routinely analysed for Gram stain, Bacterial culture & sensitivity, AFB smear, and culture & sensitivity. These values were noted and analysed for their relation with outcome.

Imaging

Though CT and MRI were seen in some cases, Contrast enhanced CT of the brain was taken for the study and any available MRI was used to confirm the findings in the CT. CT scan of all the patients with hydrocephalus were studied and looked for gyral enhancement and infarcts. They were individually studied for their relation with the outcome of these patients

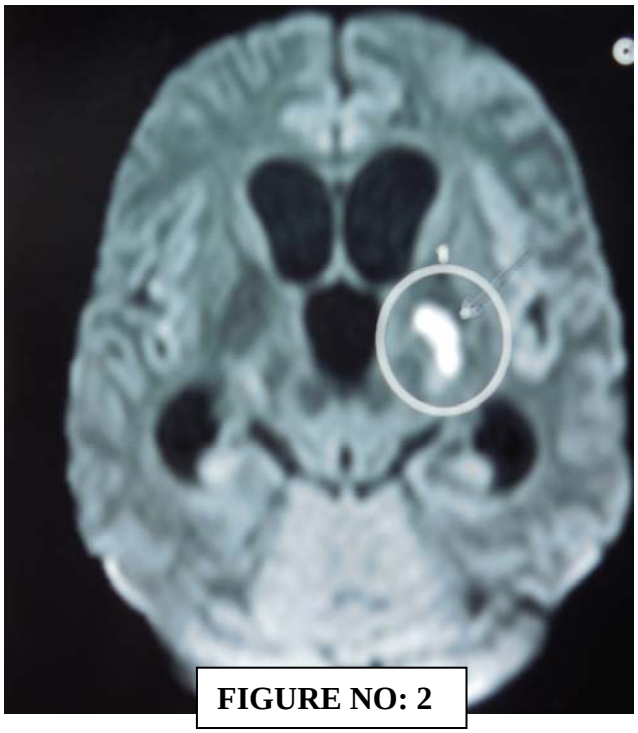


FIGURE NO: 2

Grading

The modified Vellore grading[40] was applied to all the patients studied and its relationship and relevance to the outcome was analysed.

Time interval for surgery

The interval for the patient to undergo shunt surgery from the time of admission was reviewed and patients were grouped into those who had surgery before 48 hrs and those who underwent surgery after 48 hrs[69]. The two groups were analysed separately for their outcome.

Outcome

All the data for a period of three months was analysed and outcome was measured. Patients were classified into three groups namely

- Recovery – patients recover completely with no residual neurological deficits
- Death – patients die within this three months
- Sequelae – all the patients in between the above mentioned groups and includes residual cranial nerve palsies, hemiparesis, hemiplegias, persistent vegetative state

Parameters including clinical, historical and imaging data were taken for all the 65, patients (as per the enclosed proforma) and their relationship with outcome was analysed. All of them had undergone ventriculoperitoneal shunt surgery.

Methods of statistical analysis

All the data was analysed using epi info software to calculate the frequency distribution and SPSS version 15 for windows for calculating the p value and taking p value of <0.05 as significant.

OBSERVATIONS AND RESULTS

The overall results of this study are as shown below

1) Age distribution of the patients

AGE of the patient	Number of Patients
2 months	10
3 months	5
4 months	4
5 months	5
6 months	1
7 months	6
8 months	1
11 months	3
1 yr	4
1.5 yrs	2
2 yrs	5
3 yrs	1
3.5 yrs	2
4 yrs	1
4.5 yrs	1
5 yrs	4
6 yrs	4
7 yrs	2
9 yrs	1
11yrs	1
12 yrs	1
14 yrs	1

TABLE NO: 1

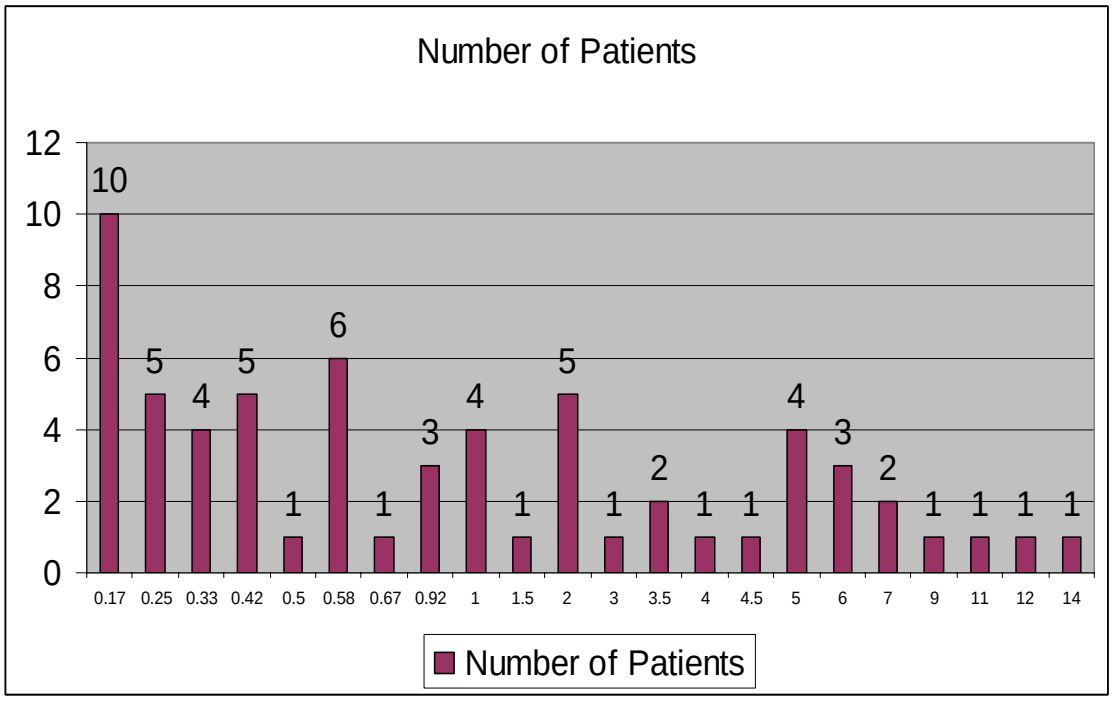


FIGURE NO: 3

As seen above, among the pediatric cases of post meningitic hydrocephalus studied, there is a small albeit a significant number of patients in neonatal period and infancy.

2) Sex Distribution

Sex distribution of the study population is as shown below

ANALYSIS OF SEX DISTRIBUTION

SEX	Number of Patients	Percent
Male	32	49.23%
Female	33	50.77%
Total	65	100.00%

TABLE NO: 2

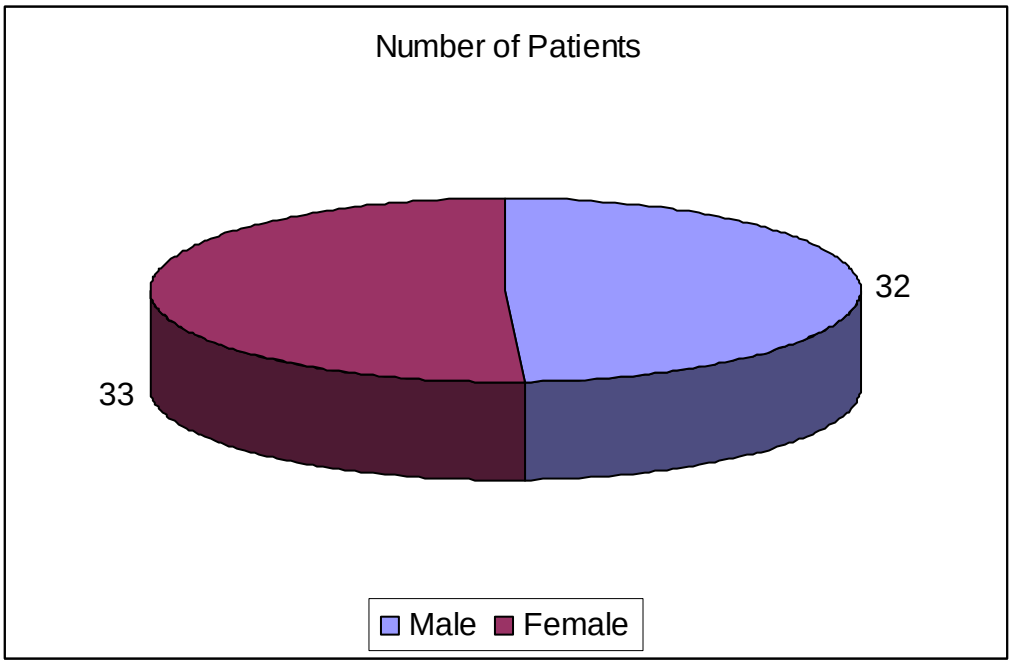


FIGURE NO:4

As can be seen in the above table and figure the sex distribution of the study population is almost equal with males comprising 49.23% and females contributing to the rest of the 50.77% of the group.

3) Fever & Duration

All the patients had fever but the days of fever was varying and the duration of illness was analysed.

Days of fever is correlated with no of patients and tabulate and charted below.

Analysis of duration of fever in days and the number of patients

DURATION OF FEVER IN DAYS	Number of Patients
7	1
8	2
9	1
14	2
15	1
16	3
17	7
18	5
19	3
20	1
21	2
22	7
23	3
24	5
25	5
26	5
27	2
28	4
29	6
Total	65

TABLE NO: 3

Statistics : Duration of Fever	
Mean	21.37
Std. Error of Mean	.688
Median	22.00
Mode	17(a)
Std. Deviation	5.547
Variance	30.768
Skewness	-.656
Std. Error of Skewness	.297
Range	22
Minimum	7
Maximum	29
Multiple modes exist. The smallest value is shown	

TABLE NO:4

The duration of fever in the patients varied from a minimum of 7 days upto 29 days with a mean of 21.37 and a standard deviation of 5.547.

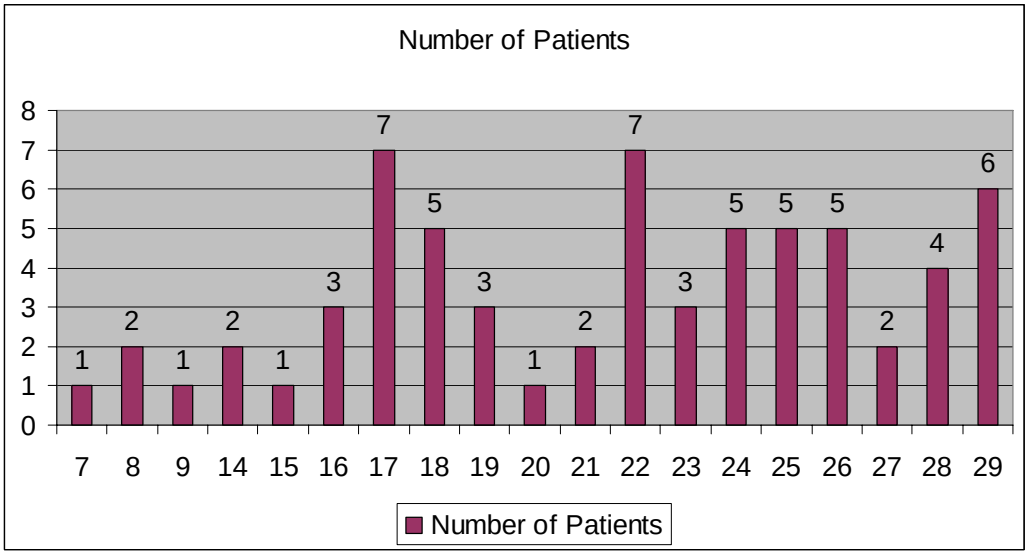


FIGURE NO: 5

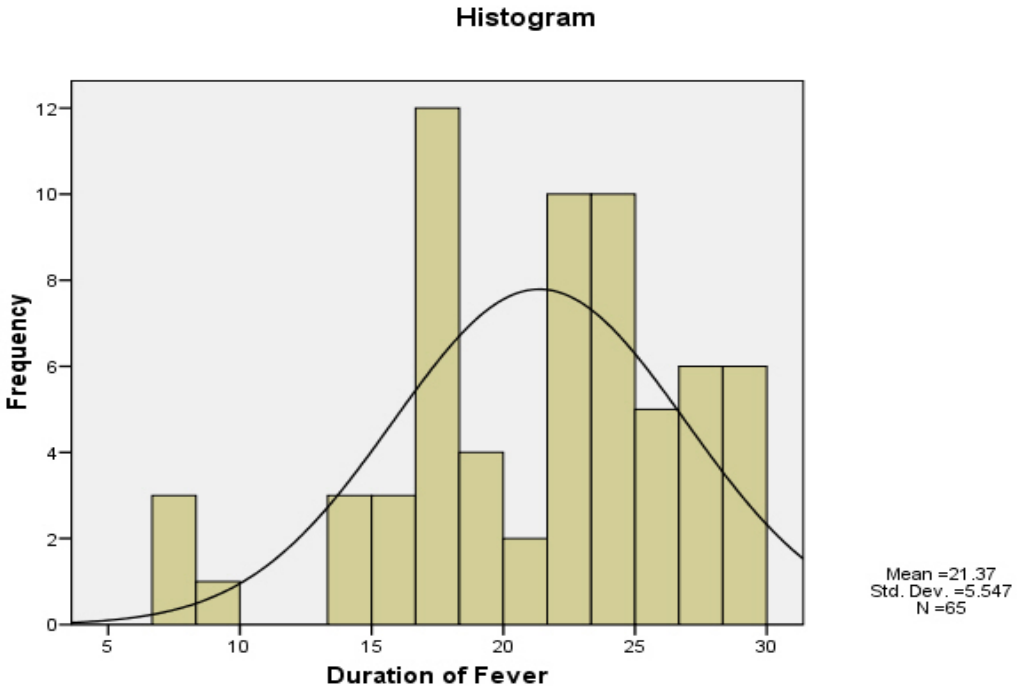


FIGURE NO: 6

As can be seen with the above histogram there is a bimodal distribution of the fever with a skew to the right, this can be accounted for the major no of tuberculous meningitis patients in this study group with a more protracted illness than pyogenic meningitis with a shorter duration before presenting, but contributing a lesser number.

4) GCS on Admission

GCS on Admission	Number of Patients	Percentage
8 and Less than 8	13	20.0
9 to 14	18	27.7
15	34	52.3
Total	65	100

TABLE NO: 5

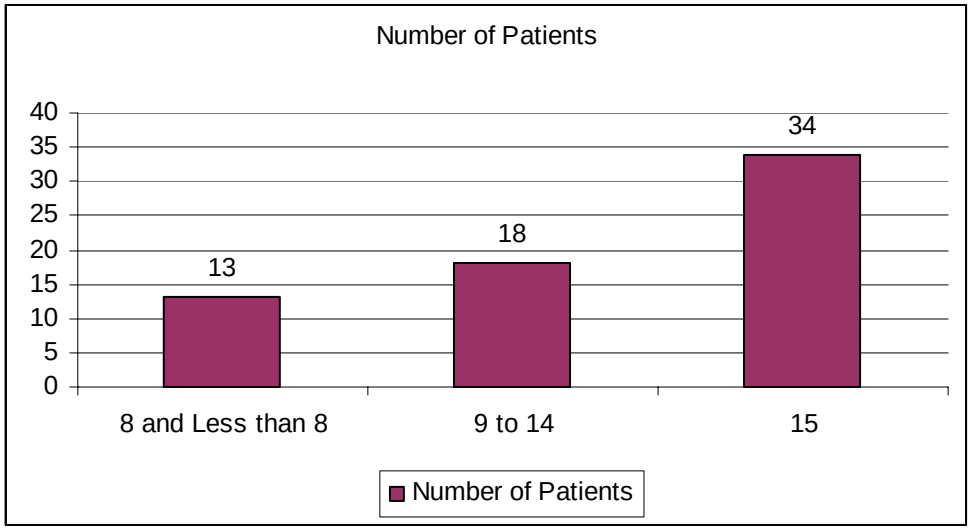


FIGURE NO: 7

The study group has been divide into three based on GCS taking into consideration the modified vellore grading[40], as mentioned previously and the majority of the group fell into GCS 15 (52.3%) followed by those between 9 and 14 (27.7%), and lastly those with GCS 8 and less (20%).

5) SEIZUZRES ON ADMISSION

SEIZURES	Number of Patients	Percent
No Seizures	43	66.15%
Seizures	22	33.85%
Total	65	100.00%

TABLE NO: 6

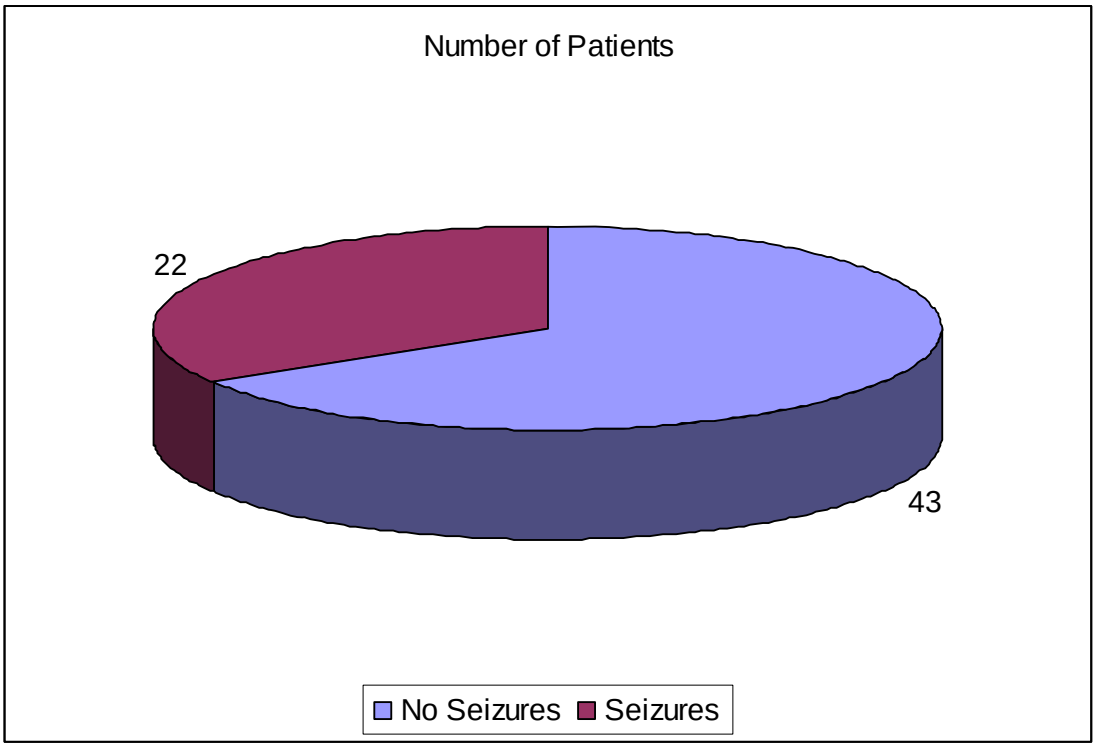


FIGURE NO: 8

In our study group 22 patients (33.85%) had seizures and the rest 43 patients (66.15%) did not have seizures.

6) FOCAL NEUROLOGICAL DEFICITS

FOCAL DEFICITS	Number of Patients	Percent
No Focal Deficits	37	56.92%
Focal Deficits	28	43.08%
Total	65	100.00%

TABLE NO: 7

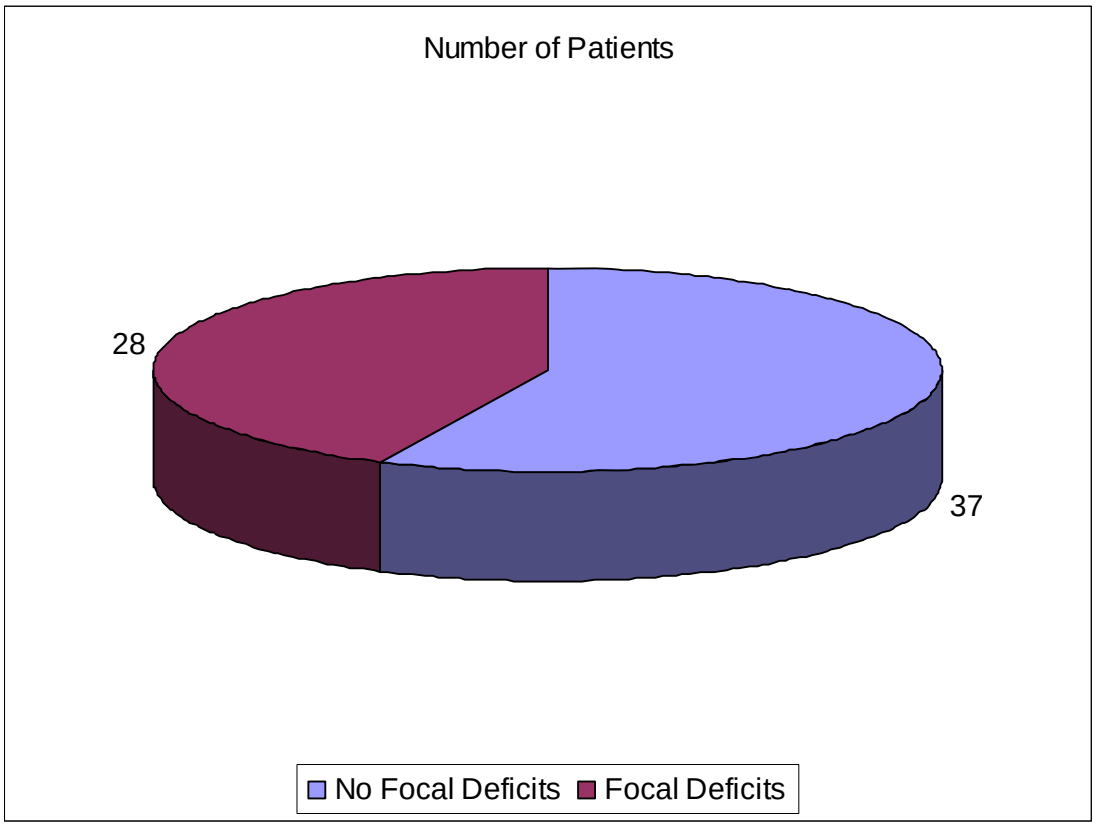


FIGURE NO: 9

Of all the 65 patients in this study group a total 28 patients (43.08%) had focal deficits including single cranial palsies, hemiparesis or hemiplegia or multiple neurological deficits, the rest 37 patients (56.92%) had no focal neurological deficits.

7) CT SCAN FINDINGS

A) *EXUDATES ON CT SCAN*

EXUDATES IN CT SCAN	Number of Patients	Percent
No Exudates in CT Scan	23	35.38%
Exudates in CT Scan	42	64.62%
Total	65	100.00%

TABLE NO: 8

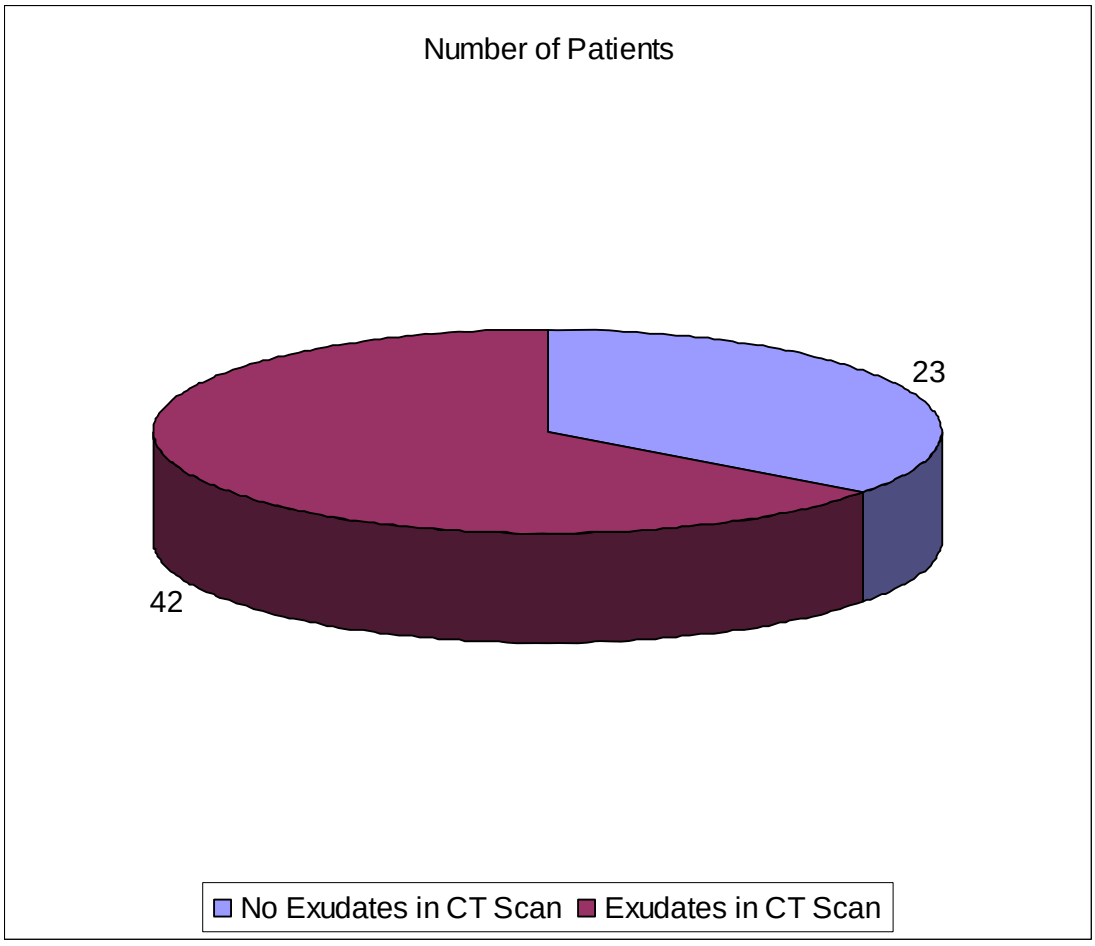


FIGURE NO: 10

A total of 42 patients (64.62%) had exudates in the basal or sylvian region and the rest 23 patients (35.38%) had no exudates on CT brain.

B) ***INFARCTS IN CT BRAIN***

INFARCTS IN CT SCAN	Number of Patients	Percent
No Infarcts in CT Scan	54	83.08%
Infarcts in CT Scan	11	16.92%
Total	65	100.00%

TABLE NO: 9

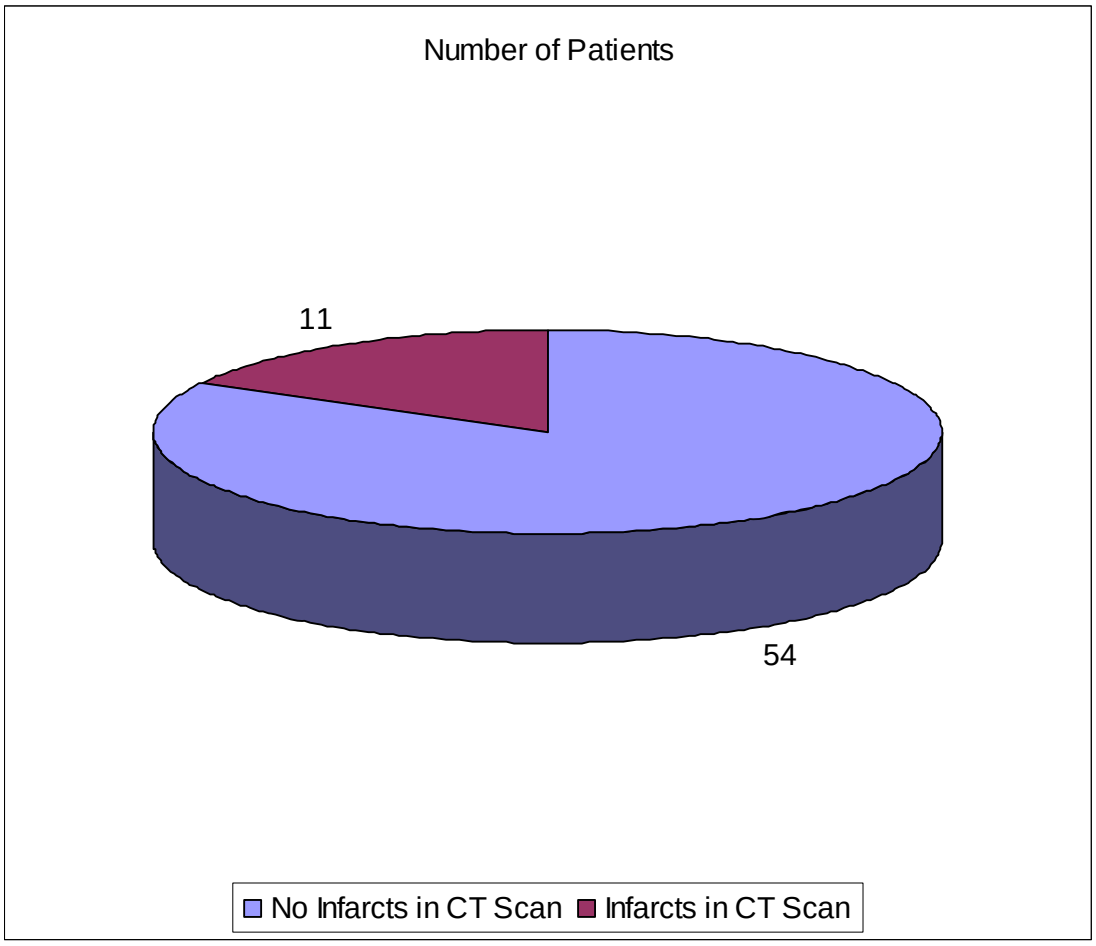


FIGURE NO: 11

Of all the patients analysed 11 patients (16.92%) had infarcts in and around the basal ganglia region and the rest 54 patients (83.08%) had no infarcts on imaging.

C) TUBERCULOMA ON CT SCAN BRAIN

TUBERCULOMA IN CT SCAN	Number of Patients	Percent
No Tuberculoma in CT Scan	58	89.23%
Tuberculoma in CT Scan	7	10.77%
Total	65	100.00%

TABLE NO: 10

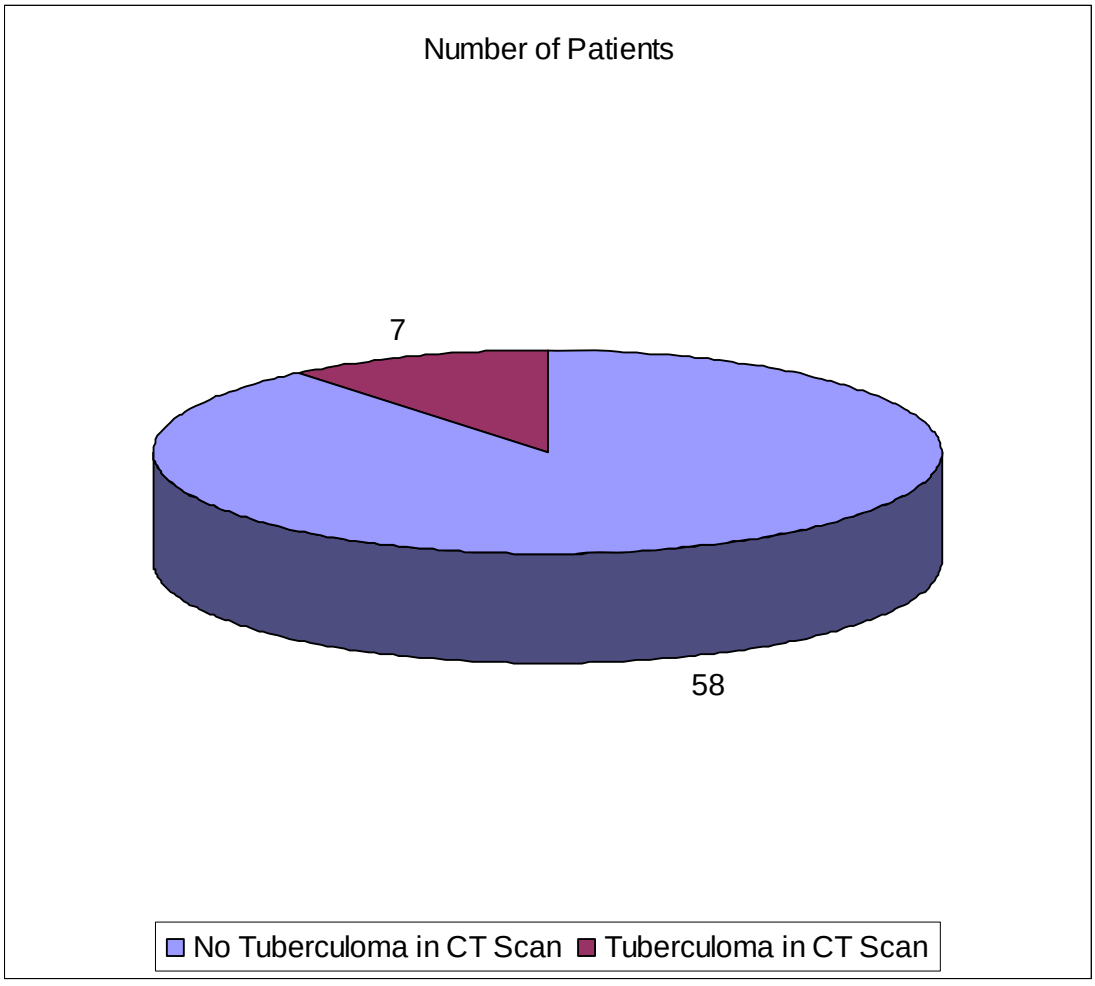


FIGURE NO: 12

A total of 7 patients (10.77%) had a single or multiple tuberculomas, the rest 58 patients (89.23%) had no tuberculomas on imaging.

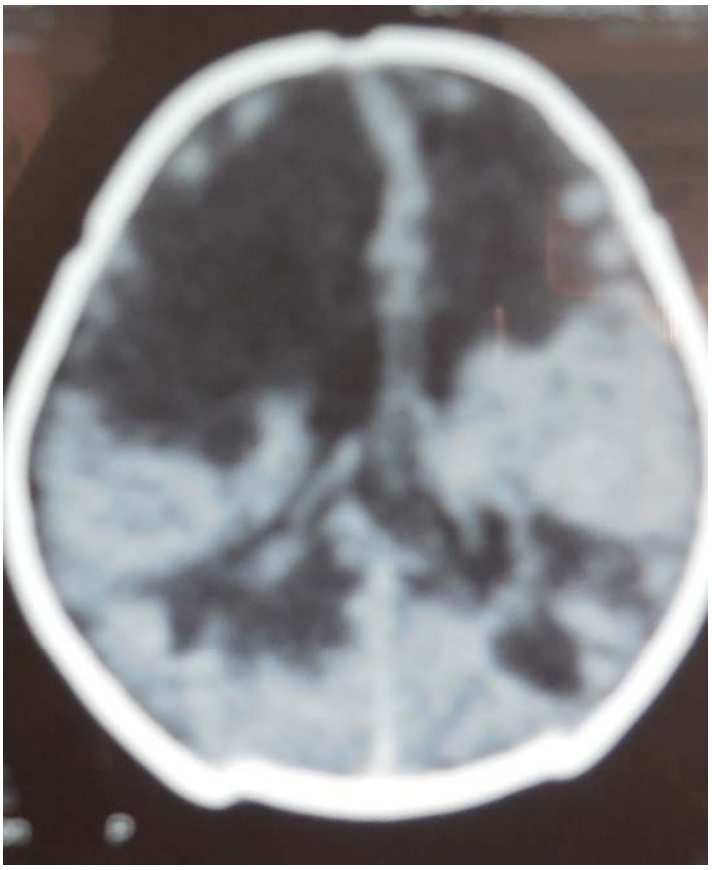


FIGURE NO: 13

FIGURE NO: 14

8) TIME INTERVAL FOR SURGERY

TIME INTERVAL FOR SURGERY	Number of Patients	Percent
Time Interval < 48 hours after admission	43	66.15%
Time Interval > 48 hours after admission	22	33.85%
Total	65	100.00%

TABLE NO: 11

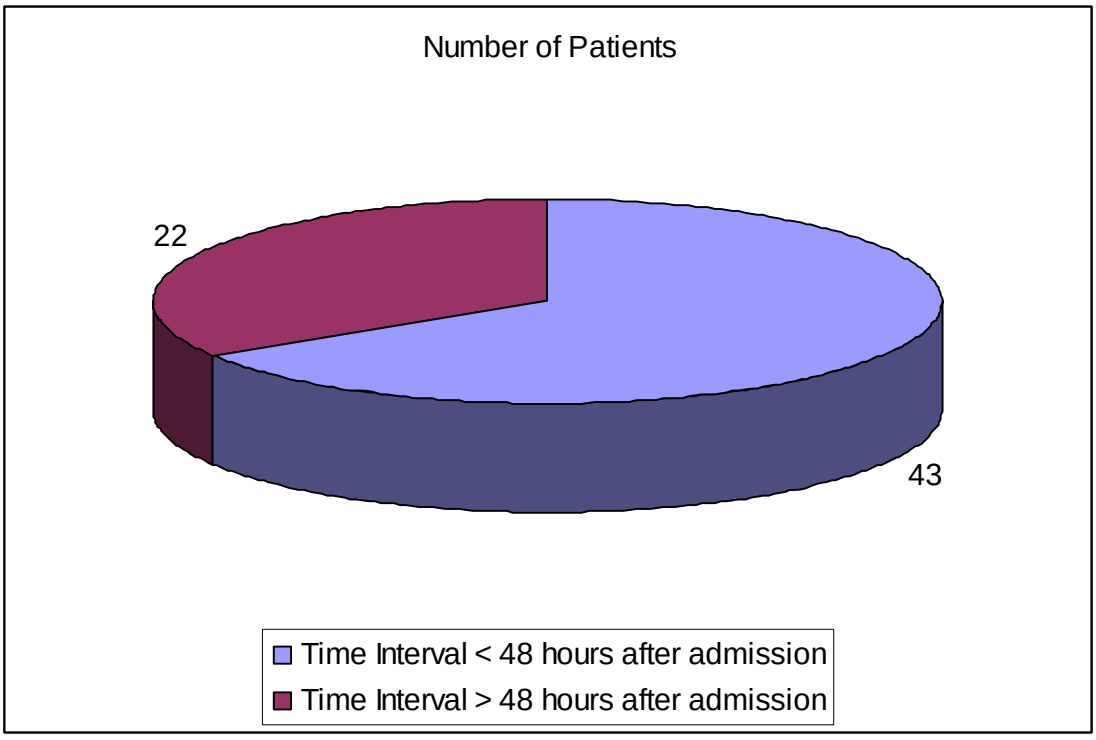


FIGURE NO: 15

As seen above 43 patients (66.15%) had undergone shunt surgery for hydrocephalus within 48 hours of admission and the rest 22 patients (33.85%) had undergone surgery after 48 hours for various reasons like confounding diagnosis, poor general condition to undergo surgery etc.

9) CSF ANALYSIS

A) CSF LYMPHOCYTOSIS

CSF LYMPHOCYTOSIS	Number of Patients	Percent
No Lymphocytosis in CSF	9	13.85%
Lymphocytosis in CSF	56	86.15%
Total	65	100.00%

TABLE NO: 12

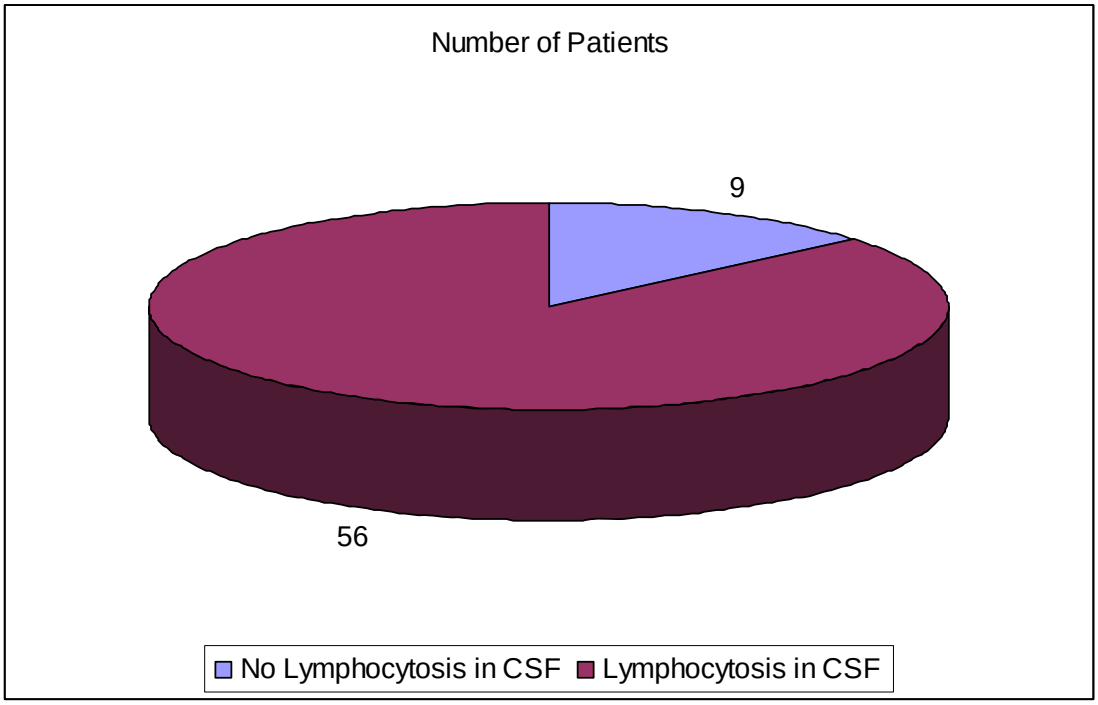


FIGURE NO: 16

CSF lymphocyte predominance was seen in 56 patients (86.15%) and the rest 9 patients (13.85%) had a neutrophil predominance

B) ***CSF PROTEIN***

STATISTICAL ANALYSIS OF CSF PROTEINS

	CSF Protein
Mean	302.42
Std. Error of Mean	21.087
Median	276.00
Mode	107(a)
Std. Deviation	170.012
Variance	28903.965
Skewness	1.017
Std. Error of Skewness	.297
Range	860
Minimum	60
Maximum	920

TABLE NO: 13

The collected data on CSF protein for the 65 patients is analysed for the range The protein values in CSF had a minimum of 60 gm/dl and a maximum of 920 gm/dl with a wide range of 860. The mean CSF protein was 302.42 mg/dl with a standard deviation of 170.012.

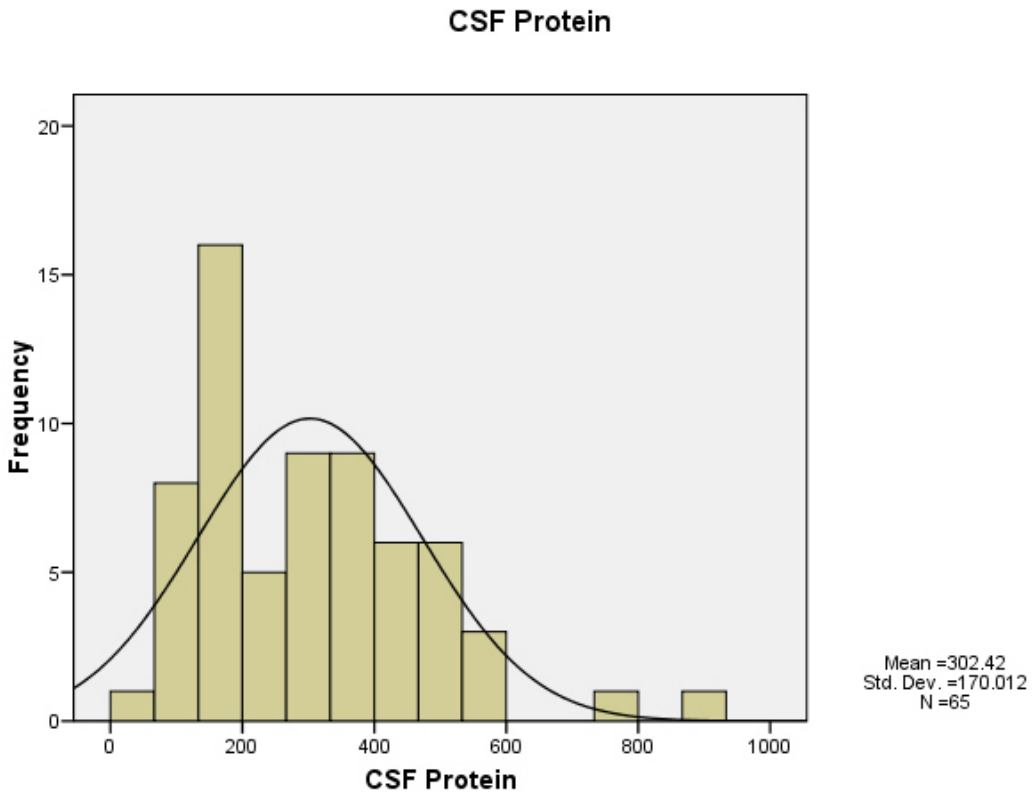


FIGURE NO: 17

As can be seen above there is a tendency of the values to clump in the range of 120 to 500 and thus the skew to the left.

CSF PROTEIN	Number of Patients	Percent
CSF Protein <200	25	38.46%
CSF Protein >200	40	61.54%
Total	65	100.00%

TABLE NO: 14

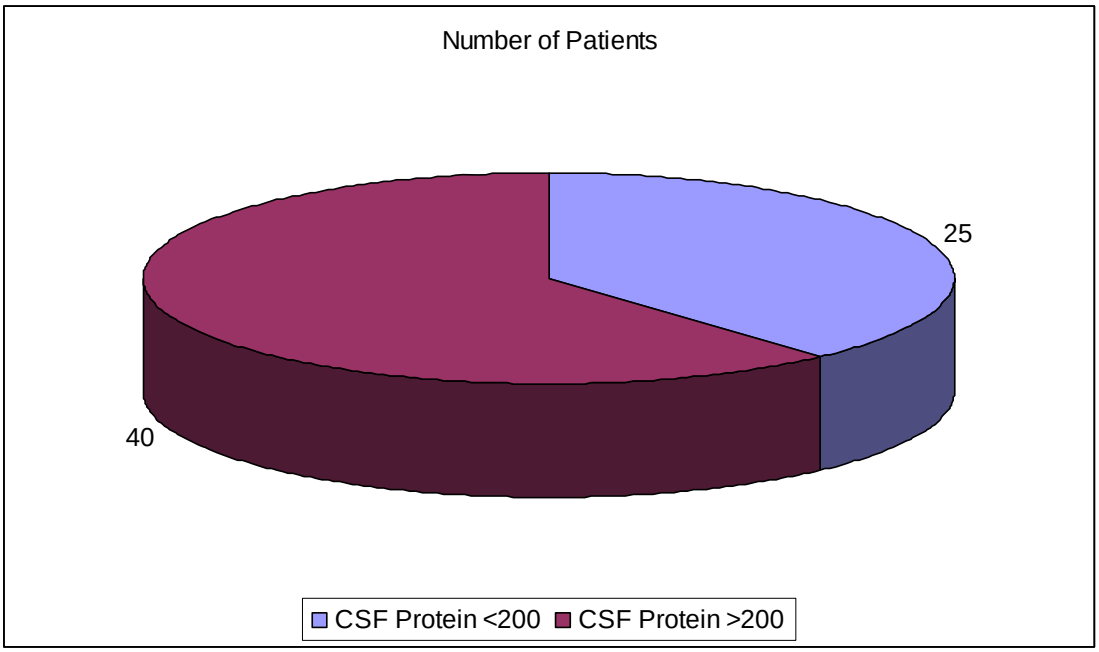


FIGURE NO:18

Patients were grouped into two and 40 patients (61.54%) had a CSF protein of more than 200gm/dl and the rest 25 patients (38.46%) had a CSF of less than 200 gm/ dl but still more than 60 gm/dl.

*C) **CSF SUGAR***

In the study population, the percentage of CSF sugar when compared to the serum sugar master chart had a minimum value of 12% and a maximum value of 60% with a range of 48. The mean was 40.86 with a standard deviation of 10.338.

	CSF Sugar
Mean	40.86
Std. Error of Mean	1.282
Median	42.00
Mode	43
Std. Deviation	10.338
Variance	106.871
Skewness	-.155
Std. Error of Skewness	.297
Range	48
Minimum	12
Maximum	60

TABLE NO: 15

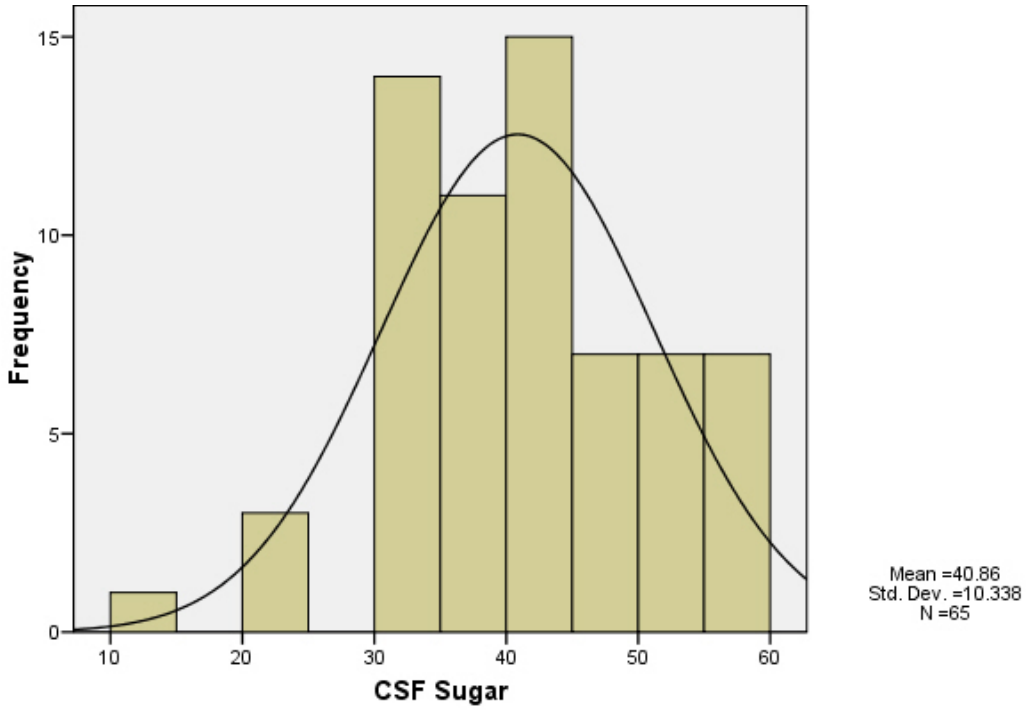


FIGURE NO: 19

The histogram reveals more patient to the right of the mean indicating that predominance in the area of 40%.

*D) **CSF GRAM STAIN***

CSF GRAM STAIN	Number of Patients	Percent
CSF GRAM STAIN Negative	56	86.15%
CSF GRAM STAIN Positive	9	13.85%
Total	65	100.00%

TABLE NO: 16

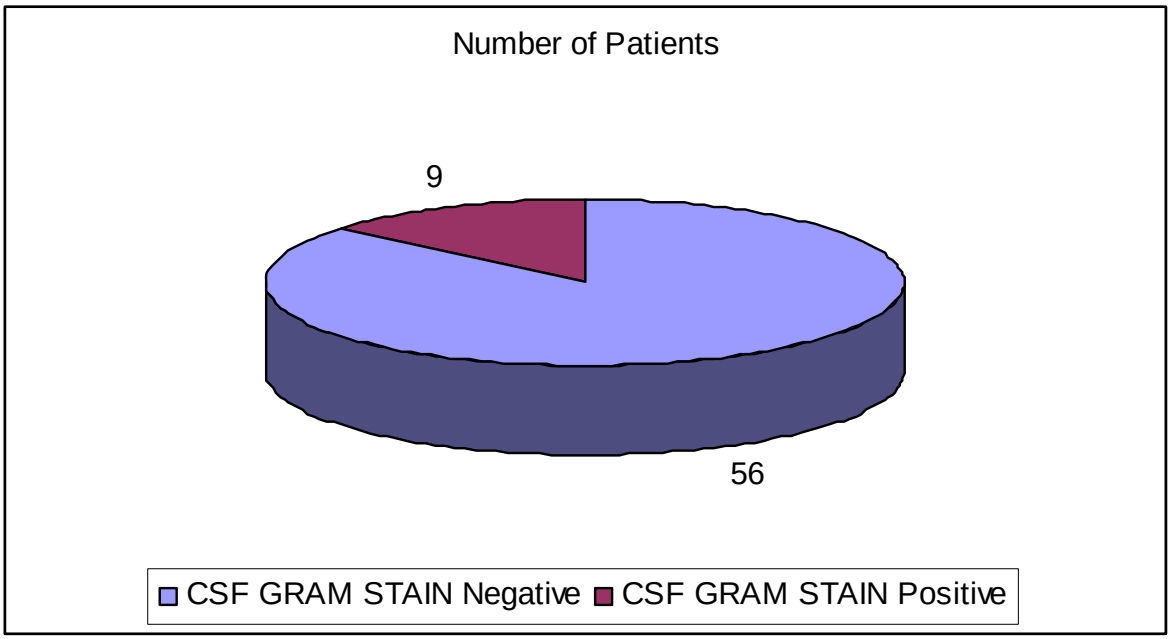


FIGURE NO: 20

In the studied group 9 patients (13.85%) showed gram stain positivity on CSF analysis and the rest 56 patients (86.15%) showed no gram staining organisms in the CSF.

E) CSF AEROBIC C/S

CSF AEROBIC C/S	Number of Patients	Percent
CSF AEROBIC C/S Negative	60	92.31%
CSF AEROBIC C/S Positive	5	7.69%
Total	65	100.00%

TABLE NO: 17

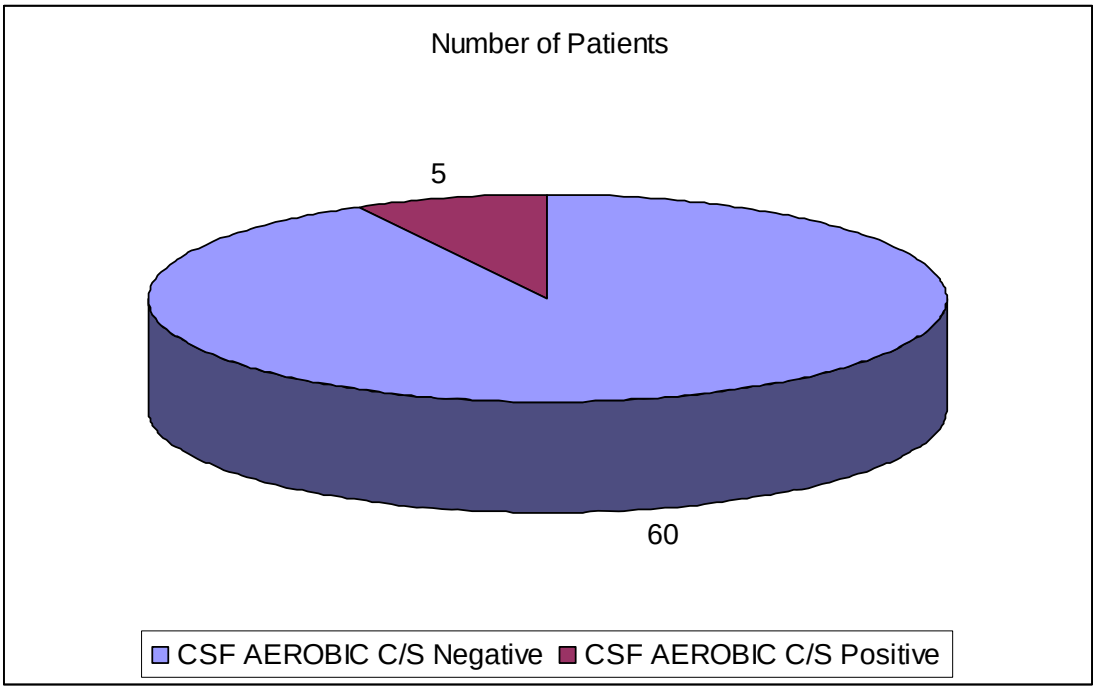


FIGURE NO: 21

Of all the patients sent for CSF aerobic C/S 5 (7.69%) of them showed growth, the rest did not show any growth on CSF aerobic C/S. None of he patients had grown any organism on anerobic culture & sensitivity of the CSF.

F) CSF AFB STAIN

CSF AFB STAIN	Number of Patients	Percent
CSF AFB STAIN Negative	57	87.69%
CSF AFB STAIN Positive	8	12.31%
Total	65	100.00%

TABLE NO: 18

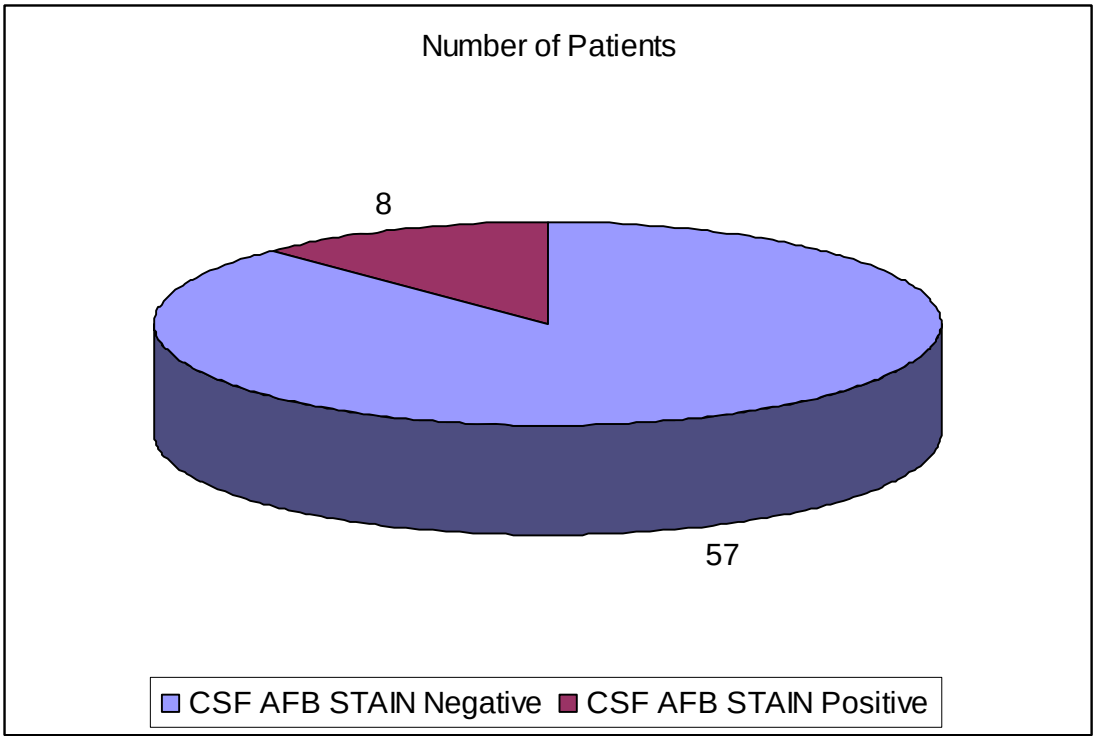


FIGURE NO: 22

CSF AFB had showed positivity in 8 patients (12.31%) , 57 patients (87.69%) did not show any AFB staining pathogens in CSF.

G) CSF AFB C/S

CSF AFB C/S	Number of Patients	Percent
CSF AFB C/S Negative	64	98.46%
CSF AFB C/S Positive	1	1.54%
Total	65	100.00%

TABLE NO: 19

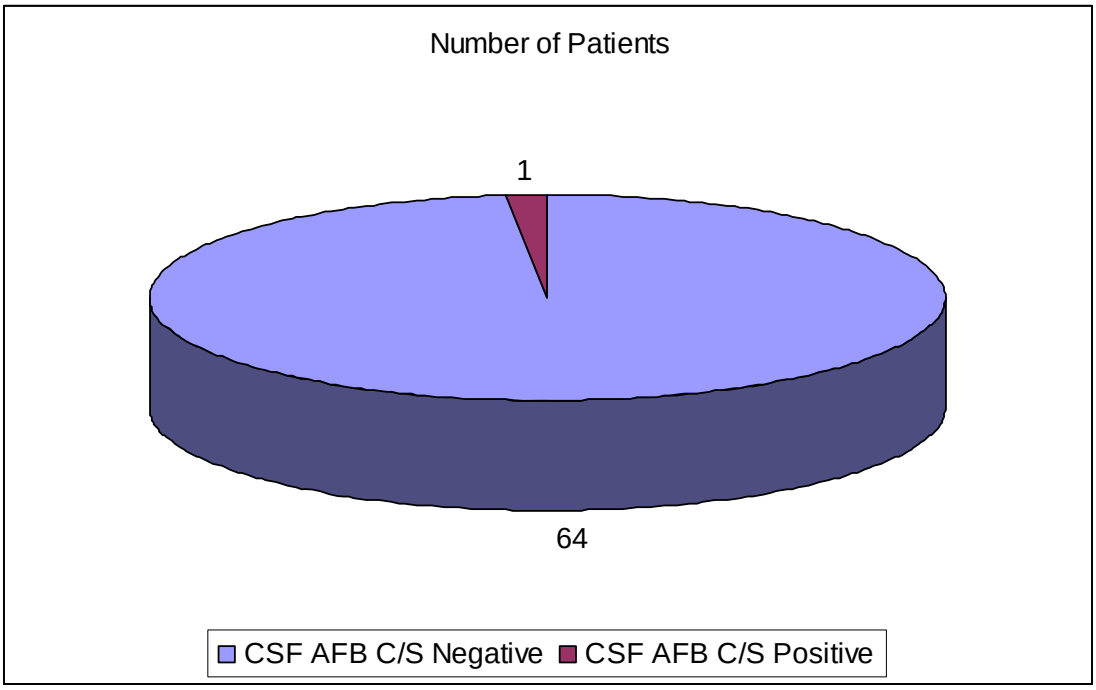


FIGURE NO: 23

One patient (1.54%) in the studied group showed growth on AFB culture sensitivity, all the other showed negative culture. There was no growth observed in fungal culture & sensitivity in any of the patients studied.

10) MODIFIED VELLORE GRADING

ANALYSIS OF MODIFIED VELLORE GRADING AND ITS DISTRIBUTION

VELLORE GRADE	Number of Patients	Percent
VELLORE GRADE 1	20	30.77%
VELLORE GRADE 2	14	21.54%
VELLORE GRADE 3	18	27.69%
VELLORE GRADE 4	13	20.00%
Total	65	100.00%

TABLE NO: 20

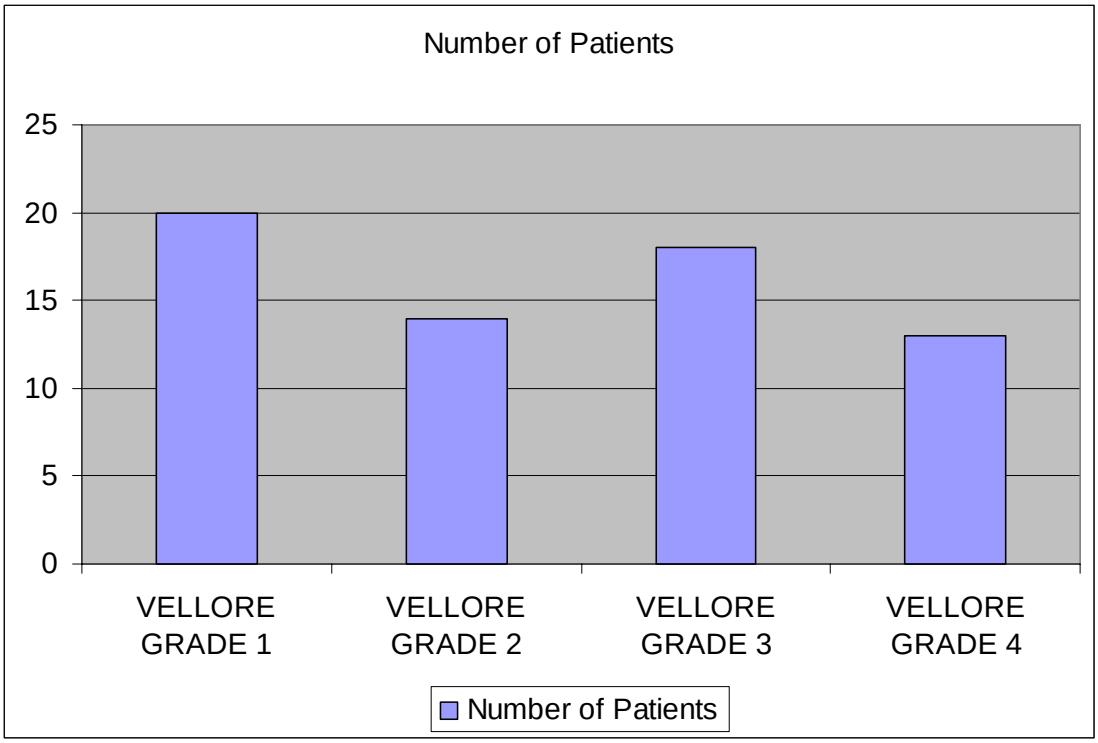


FIGURE NO: 24

There was an almost equal percentage of patients distributed among the four grades with 20 patients (30.77%) in grade 1, 14 patients (21.54%) in grade 2, 18 patients (27.69%) in grade 3 and 13 patients (20%) in grade 4.

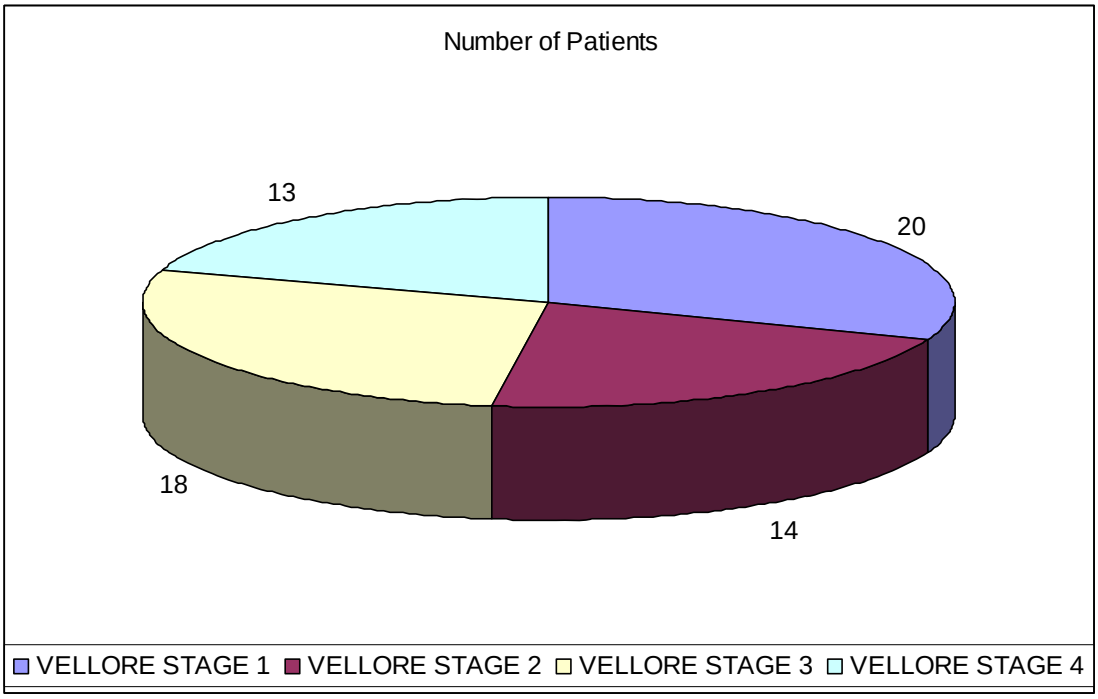


FIGURE NO: 25

The above figure shows an almost equal distribution of patients among all the grades.

19) **OUTCOME**

OUTCOME	Number of Patients	Percent
Death	18	27.69%
Recovery	32	49.23%
Sequelae	15	23.08%
Total	65	100.00%

TABLE NO: 21

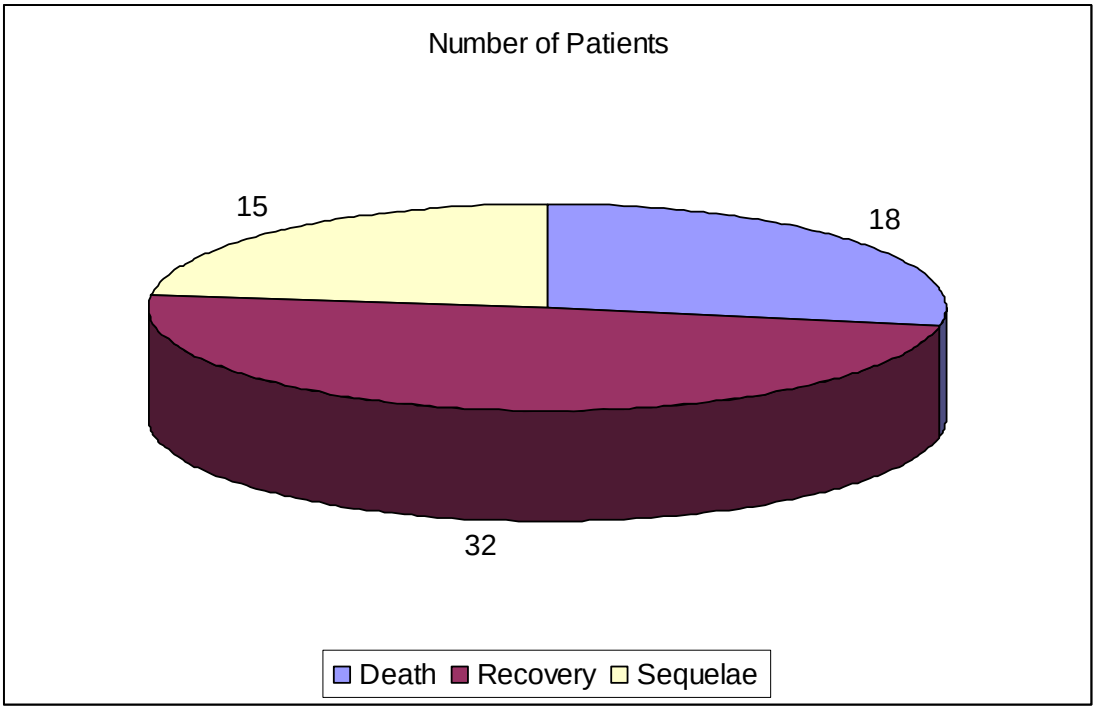


FIGURE NO: 26

In total, 18 of the patients (27.69%) studied had died, 32 (49.23%) had completely recovered and 15 patients (23.08%) had neurological sequelae at the end of three months.

ANALYSIS AND DISCUSSION

In total 65 patients were taken and studied for various parameters as seen above, out of the 65 patients, 9 of them were pyogenic meningitis and the rest were due to tuberculous meningitis.

In this study when age is analysed, 25 children were < 6 months of age, while the rest 40 children were between 6 months of age and 14 years of age.

The study population had 33 female children and 32 male children, without almost both the sexes contributing equally.

Among the study group only 7 children had fever for 2 weeks or less, all the other children had a preotracted illness lasting for more than 2 weeks and lasting upto 1 month.

Further analysis of the data was done using SPSS software to calculate the p value for their statistical significance.

1) RELATIONSHIP BETWEEN GCS ON ADMISSION AND OUTCOME

	Outcome			
GCS ON ADMISSION	**Death**	**Recovery**	**Sequelae**	**Total**
8	9	2	2	13
Row%	69.23%	15.38%	15.38%	100.00%
Col%	50.00%	6.25%	13.33%	20.00%
9 to 14	5	7	6	18
Row%	27.78%	38.89%	33.33%	100.00%
Col%	27.78%	21.88%	40.00%	27.69%
15	4	23	7	34
Row%	11.76%	67.65%	20.59%	100.00%
Col%	22.22%	71.88%	46.67%	52.31%
TOTAL	18	32	15	65
Row%	27.69%	49.23%	23.08%	100.00%
Col%	100.00%	100.00%	100.00%	100,00%
P value = 0.0011				

TABLE NO: 22

On analysis of the GCS on admission along with outcome, hence individual outcomes were correlated with GCS on admission.

p value for

GCS and death – 0.0004

GCS and recovery -0.0034

GCS and sequeqle – 0.4452

On detailed analysis of the GCS on admission and its relation to the outcome as death, recovery or sequelae it was found to have a significant association with predicting death and recovery with the p value for death as outcome being 0.0004 and that for recovery being 0.0034. Analysis of this study correlates well with many previous studies in implying the relationship of poor GCS to death [12, 15, 69],[56, 70,71, 72, 73, 74] and a good GCS on admission with recovery and GCS not being a good predictor of sequelae.

2) RELATIONSHIP BETWEEN SEIZURE AND OUTCOME

In this study 22 children had seizures, while 43 children did not have seizures.

ANALYSIS OF SEIZURE AND OUTCOME

	Outcome			
SEIZURES	**Death**	**Recovery**	**Sequelae**	**Total**
NO	4	29	10	43
Row%	9.30%	67.44%	23.26%	100.00%
Col%	22.22%	90.63%	66.67%	66.15%
YES	14	3	5	22
Row%	63.64%	13.64%	22.73%	100.00%
Col%	77.78%	9.38%	33.33%	33.85%
TOTAL	18	32	15	65
Row%	27.69%	49.23%	23.08%	100.00%
Col%	100.00%	100.00%	100.00%	100.00%
p value = 0				

TABLE NO: 23

p value for

seizure and death – 0.000015

seizure and recovery – 0.00012

seizure and sequelae – 0.792

On analysis of the presence of seizures to the outcome, it was found to have a good correlation in predicting death and recovery with respective p values of 0.000015 and 0.00012 which implies that the occurrence of seizure predicts a relative higher number of deaths and conversely, their absence predicting recovery, with no effect on sequelae in three months. This compares well with *Hosoglu* et al[12] and *Paganini*[75] et al. The probable explanation is patients with seizures might be having more extensive cerebral damage than those who don't have it, consequently the poor outcome

3) RELATIONSHIP BETWEEN FOCAL DEFICIT AND OUTCOME

In this study, a total of 28 patients had focal neurological deficits, the rest 37 patients did not have any demonstrable deficit.

ANALYSIS OF FOCAL DEFICIT AND OUTCOME

	Outcome			
FOCAL DEFICITS	**Death**	**Recovery**	**Sequelae**	**Total**
NO	8	26	3	37
Row%	21.62%	70.27%	8.11%	100.00%
Col%	44.44%	81.25%	20.00%	56.92%
YES	10	6	12	28
Row%	35.71%	21.43%	42.86%	100.00%
Col%	55.56%	18.75%	80.00%	43.08%
TOTAL	18	32	15	65
Row%	27.69%	49.23%	23.08%	100.00%
Col%	100.00%	100.00%	100.00%	100.00%
p value = 0.0002				

TABLE NO: 24

p value for

focal deficit and death – 0.32

focal deficit and recovery – 0.0002

focal deficit and sequelae – 0.002

Analysing the data on the occurrence of focal deficits it was found to have a significant influence on recovery and sequelae with a p value of 0.0002 and 0.002 implying that patients without focal deficits have a complete recovery and those with deficits land up in sequelae. The occurrence of focal deficits does not have any influence on death of the patients. These observations are similar to that of *J. Kalita*[71] et al who maintain that focal deficit is the single most important predictor of sequelae in these patients.

4) RELATIONSHIP BETWEEN VELLORE GRADE AND OUTCOME

Applying the modified vellore grading on the study sample on meningitis with hydrocephalus patients, the following results are obtained.

ANALYSIS OF VELLORE GRADE AND OUTCOME

	Outcome			
VELLORE GRADE	**Death**	**Recovery**	**Sequelae**	**Total**
1	1	17	2	20
Row%	5.00%	85.00%	10.00%	100.00%
Col%	5.56%	53.13%	13.33%	30.77%
2	3	6	5	14
Row%	21.43%	42.86%	35.71%	100.00%
Col%	16.67%	18.75%	33.33%	21.54%
3	5	7	6	18
Row%	27.78%	38.89%	33.33%	100.00%
Col%	27.78%	21.88%	40.00%	27.69%
4	9	2	2	13
Row%	69.23%	15.38%	15.38%	100.00%
Col%	50.00%	6.25%	13.33%	20.00%
TOTAL	18	32	15	65
Row%	27.69%	49.23%	23.08%	100.00%
Col%	100.00%	100.00%	100.00%	100.00%
p value = 0.0004				

TABLE NO: 25

p value for modified vellore grading and death – 0.0008, while p value for modified vellore grading and recovery – 0.0006 while p value for modified vellore grading and sequelae – 0.1963

On analysis of the grading of the patients to the outcome it was found to have a statistical significance in predicting death and recovery with a p value of 0.0008 and 0.0006 with no significant association with predicting sequelae, implying complete recovery for a better grade patient and death for a poor grade patient. These observations have been ratified previously by many authors, *Palur et al*[76] , *Mathew et al*[40], *Agarwal et al*[10], *Singh and kumar et al*[77].

5) ANALYSIS OF THE RELATION BETWEEN CT FINDINGS AND THE OUTCOME

A) RELATIONSHIP BETWEEN INFARCTS ON CT SCAN BRAIN AND OUTCOME

A total of 11 patients were found to have infarcts in their CT brain and they were analysed.

ANALYSIS OF INFARCTS IN CT BRAIN AND OUTCOME

	Outcome			
INFARCTS IN CT SCAN	**Death**	**Recovery**	**Sequelae**	**Total**
NO	16	27	11	54
Row%	29.63%	50.00%	20.37%	100.00%
Col%	88.89%	84.38%	73.33%	83.08%
YES	2	5	4	11
Row%	18.18%	45.45%	36.36%	100.00%
Col%	11.11%	15.63%	26.67%	16.92%
TOTAL	18	32	15	65
Row%	27.69%	49.23%	23.08%	100.00%
Col%	100.00%	100.00%	100.00%	100.00%
p value = 0.4762				

TABLE NO: 26

p value for

CT infarcts and death - 0.68

CT infarct and revovery – 0.95

CT infarct and sequelae – 0.45

In this study, infarcts in CT brain has been found to have no correlation with outcome, this is in contrast to *Sil and Chatterjee*[78], but the above finding has already been questioned by *Rajasekhar et al*[79] as it can be a confounding factor , in that more infarcts might have been observed in poor grade patients. Our conclusion is similar to *Kingsley et al*[80] who found infarcts on CT to have no influence in outcome of the patient.

B) RELATIONSHIP BETWEEN EXUDATES IN CT SCAN BRAIN AND OUTCOME

ANALYSIS OF EXUDATES IN CT BRAIN AND OUTCOME

	Outcome			
EXUDATES IN CT SCAN	**Death**	**Recovery**	**Sequelae**	**Total**
NO	5	12	6	23
Row%	21.74%	52.17%	26.09%	100.00%
Col%	27.78%	37.50%	40.00%	35.38%
YES	13	20	9	42
Row%	30.95%	47.62%	21.43%	100.00%
Col%	72.22%	62.50%	60.00%	64.62%
TOTAL	18	32	15	65
Row%	27.69%	49.23%	23.08%	100.00%
Col%	100.00%	100.00%	100.00%	100.00%
p value = 0.7197				

TABLE NO: 27

p value for

CT exudates and death – 0.61

CT exudate and recovery – 0.92

CT exudate and sequelae – 0.90

Basal exudates on CT brain was found to have no relationship to the outcome of the patient, as seen by *Kingsley et al*[80].

6) RELATIONSHIP BETWEEN TIME AND INTERVAL FOR SURGERY AND OUTCOME

43 patients underwent shunt surgery in less than 48 hours while 22 patients underwent surgery after 48 hours after admission.

ANALYSIS BETWEEN TIME INTERVAL FOR SURGERY AND OUTCOME

	Outcome			
TIME INTERVAL FOR SURGERY	**Death**	**Recovery**	**Sequelae**	**Total**
<48	6	27	10	43
Row%	13.95%	62.79%	23.26%	100.00%
Col%	33.33%	84.38%	66.67%	66.15%
>48	12	5	5	22
Row%	54.55%	22.73%	22.73%	100.00%
Col%	66.67%	15.63%	33.33%	33.85%
TOTAL	18	32	15	65
Row%	27.69%	49.23%	23.08%	100.00%
Col%	100.00%	100.00%	100.00%	100.00%
p value = 0.012				

TABLE NO: 28

p value for

time interval for surgery and death – 0.001

time interval for surgery and recovery – 0.005

time interval for surgery and sequelae – 0.79

Time interval for surgery was found to have a significant impact on the outcome with a p value of 0.001 and 0.005 for death and recovery. It was not found to have any influence on sequelae. This has consistently been implied in many studies[40, 64, 81, 82, 83, 84, 85]

7) RELATIONSHIP BETWEEN CSF PROTEIN AND OUTCOME

ANALYSIS OF CSF PROTEIN AND OUTCOME

Analyzing the CSF protein values, 40 children had more than 200 mg/dl, while 25 children had protein of less than 200 mg/dl.

	Outcome			
CSF PROTEIN 200	**Death**	**Recovery**	**Sequelae**	**Total**
<200	5	17	3	25
Row%	20.00%	68.00%	12.00%	100.00%
Col%	27.78%	53.13%	20.00%	38.46%
>200	13	15	12	40
Row%	32.50%	37.50%	30.00%	100.00%
Col%	72.22%	46.88%	80.00%	61.54%
TOTAL	18	32	15	65
Row%	27.69%	49.23%	23.08%	100.00%
Col%	100.00%	100.00%	100.00%	100.00%
p value = 0.0154				

TABLE NO: 29

p value for

CSF protein and death – 0.41

CSF protein and recovery – 0.03

CSF protein and sequelae – 0.16

CSF protein was not found to have any influence on outcome whatever it maybe death, recovery or sequelae. This is in contrast to *Ambedkar et al*[86] who found CSF protein > 200 mg/dl had more sequelae. But many other studies were concurrent with this study.[40],[74],[76]

8) Relationship between CSF AFB positivity and outcome

Analysis Of CSF AFB Positivity And The Outcome

	Outcome			
CSF AFB STAIN	**Death**	**Recovery**	**Sequelae**	**Total**
NEGATIVE	13	30	14	57
Row%	22.81%	52.63%	24.56%	100.00%
Col%	72.22%	93.75%	93.33%	87.69%
POSITIVE	5	2	1	8
Row%	62.50%	25.00%	12.50%	100.00%
Col%	27.78%	6.25%	6.67%	12.31%
TOTAL	18	32	15	65
Row%	27.69%	49.23%	23.08%	100.00%
Col%	100.00%	100.00%	100.00%	100.00%
p value = 0.0632				

p value for

AFB stain and death – 0.05

AFB stain and recovery - 0.27

AFB stain and sequelae – 0.75

CSF for AFB stain was found to have a p value of 0.05 for death, a trend towards more significant statistically, which can probably be explained that, if the patients have demonstrable AFB on CSF the disease load might be high and henceforth the tendency towards to a grave prognosis.

CONCLUSION

1. Clinical examination with GCS of the patients with meningitis and hydrocephalus significantly predicts death and recovery
2. Seizure is also another good prognostic marker for death or recovery
3. Focal neurological deficit is a good and only prognostic marker correlating with sequelae.
4. Earlier the intervention for the hydrocephalus, better the outcome.
5. CT scan brain of the patient is a good tool in analyzing the pathological process in meningitis, but cannot be relied upon for predicting the outcome
6. Modified Vellore grading system is a good system for prognostication in children with post meningitic hydrocephalus.

BIBLIOGRAPHY

[1] PN., Tandon. Tuberculous meningitis (cranial and spinal). [book auth.] Bruyn GW Vinken PJ. Handbook of Clinical Neurology. Infections of the Nervous system Vol.33. Amsterdam: North—Holland : s.n., 1978, pp. 195-262.

[2] Toporek, Chuck, and Kellie Robinson. Hydrocephalus: A Guide for Patients, Families & Friends. Cambridge : Mass.: O'Reilly & Associates, 1999.

[3] Infantile hydrocephalus: Long term results of surgical therapy. Amacher A L, Wellington J. 1984, Childs Brain, pp. 11: 217-22

[4] Tuberculous meningitis in adults: review of 61 cases. Sutlas PN, Unal A, Forta H, Senol S and Kirbas D. 2003, Infection, pp. 31:387–91

[5] Bacterial Meningitis in Children. Susana Cha´vez-Bueno, George H. McCracken, Jr. 2005(52), Paediatric Clinics of North America, pp. 795-810

[6] The pathogenesis of tuberculous meningitis. Rich AR, McCordock HA. s.l. : Bull John Hopkins Hosp, 1933, Bull John Hopkins Hosp, pp. 52:5-37.

[7] Pathology and pathogenetic mechanisms in neurotuberculosis. Dastur DK, Manghani DK, Udani PM. 1995, Radiology Clinics North America, pp. 33: 733-752

[8] Tuberculous hydrocephalus: Comparison of different treatments with regards to ICP, ventricular size and clinical outcome. Schoeman J, Donald P, van Zyl L, Keet M, Wait J. 1991, Developmental Medicine & Child Neurology, pp. 33:396-405

[9] Neurosurgically relevant aspects of pathology and pathogenesis of intracranial and intraspinal tuberculosis. DK, Dastur. 1983, Volume 6, Issue 3, Neurosurgical Review, pp. 103-110

[10] Role of shunt surgery in paediatric tuberculous meningitis with hydrocephalus. Agarwal D, Gupta A, Mehta VS. 2005, Indian Paediatrics, pp. 24: 1029-32

[11] Tuberculosis meningitis, Abbassia Fever Hospital-Naval Medical Research Unit N0. 3-Cairo, Egypt from 1976 to 1996. Girgis NI, Sultan Y, Farid Z, Mansour MM, Erian MW, Hanna LS, et al. 1998, American Journal Tropical Medicine & Hygeine, pp. 58(1):28-34

[12] Predictors of outcome in patients with tuberculous meningitis. Hosoglu S, Geyik MF, Balik I, Aygen B, Erol S, Ayengcel TG, et al. 2002, The International Journal of Tuberculosis and lung disease, pp. 6(1):64-70.

[13] Tuberculosis of the central nervous system in children: a 20-year survey. Farinha Nj, Razai KA, Holzel H, Morgan G, Novelli VM. 2000;41(1), Journal of Infection, pp. 61-68.

[14] Tuberculous meningitis: a 30-year review. Kent SJ, Crowe SM, Yung A, Lucas CR, Mijch AM. 1993;17(6), Clinical Infectious Diseases, pp. 987-994.

[15] Tuberculous meningitis in adults: review of 48 cases. Verdon R, Chevret S, Laissy JP, Wolff M. 1996;22(6), Clinical Infectious Diseases, pp. 982-988.

[16] Tuberculous meningitis in the southwest united states: a community based study. Davis LE, Rastogi KR, LambertLC, Skipper BJ. 1993;43(9), Neurology, pp. 1775-1778.

[17] Tuberculous meningitis: many questions , too few answers. Thwaites GE, tran TH. 2005;4(3), Lancet Neurology, pp. 160-170.

[18] Central nervous system tuberculosis in children. Curless RG, Mitchell CD. 1991, Pediatric Neurology, pp. 7(4): 270-4

[19] Central nervous system tuberculosis in children: a review of 214 cases. Yaramis A, Gurkan F, Elevli M, Soker M, haspolat K, Kirbas G, et al. 1998, pediatrics, p. 102(5):E49

[20] Measurement CSF output through external ventricular drainage in one hundred infants and children. Yasuda T, Tomita T, Mc LoneDG, Donovan M. 2002;36(1), Paediatric Neurosurgery, pp. 22-28.

[21] The production of cerebrospinal fluid in man and its modification by acetazolamide. Rubin RC, Henderson ES, Ommaya AK, Walker MD, Rall DP. 1996;25(4), Journal of Neurosurgery, pp. 430-436.

[22] Supratentorial cerebrospinal fluid production rates in healthy adults: quantification with two-dimensional cine phase-contrast MR imaging with high temporal and spatial resolution. Huang TY, Chung HW, Chen MY, Giiang LH, Lee CS, et al. 2004:233(2), Radiology, pp. 603-608.

[23] Improving the bacteriological diagnosis of tuberculous meningitis. THwaites GE, Chau TT, Farrar JJ. 2004;42(1), Journal of Clinical Microbiology, pp. 378-379.

[24] 24. S.Greenberg, Mark. Handbook of Neurosurgery. Florida : Thieme, 2001.

[25] CT of the brain in tuberculous meningitis. A review of 289 patients. Ozates M, Kemaloglu S, Gurkan F, Ozkan U, Hosoglu S, Simsek MM. 200;41(1), Acta Radiologica, pp. 13-7.

[26] Clinical and laboratory characteristics of cerebral infarction in tuberculous meningitis. Koh SB, Kim BJ, Park MH, YU SW, Park KW, Lee DH. 2007;14(11), Journal of Clinical Neuroscience, pp. 1073-7

[27] Cerebral infarcts complicating tuberculosis. Chan KH, Cheung RT, Lee R, Mak W, Ho SL. 2005;19(6), Cerebrovascular Disease, pp. 391-5.

[28] Locations of cerebral infarctions in tuberculous meningitis. Hsieh FY, Chia LG, Shen WC. 1992;34(3), Neuroradiology, pp. 197-9.

[29] Serial MRI to determine the effect of dexamethasone on the cerebral pathology of tuberculous meningitis: an observational study. Thwaites GE, Macmullen-Price J, Tran TH, Pham PM, Nguyen TD, Simmons CP, et al. 2007;6(3), Lancet Neurology, pp. 230-6

[30] MRI findings of intrcranial tuberculomas. Sonmez G, Ozturk E, Sildiroglu HO, Mutlu H, Cuce F, Senol MG, et al. 2008;32(2), Clinical Imaging , pp. 88-92

[31] Brain CT and MRI findings in 100 consecutive patients with intracranial tuberculoma. Wasay M, Kheelani BA, Moolani Mk, Zaheer J, Pui M, Hasan S, et al. 2003;13(3), Journal of Neuroimaging, pp. 240-7.

[32] CT features of tuberculous intracranial abscesses in children. du Plessis J, Androniku S, WeiselthalerN, Theron S, George R, Mapukata A. 2007;37(2), Pediatric Radiology, pp. 167-72.

[33] Identification of diffuse and focal brain lesions by clinical magnetic resonance spectroscopy. Kingsley PB, ShaH TC, Woldenberg R. 2006;19(4), NMR in Biomedicine, pp. 435-62.

[34] Differential diagnosis between cerebral tuberculosis and neurocysticercosis by magnetic resonance spectroscopy. Pretell EJ, Martinot Jr C, Garcia HH, Alvarado M, Bustos JA, Martinot C. 2005;29(1), Journal of Computer Assissted Tomography, pp. 112-4

[35] 35. A diagnostic rule for tuberculous meningitis. Kumar R, Singh SN, Kohli N. 1999;81(3), Archives of Disease in Childhood, pp. 221-224.

[36] 36. Differentiation of tuberculous meningitis from acute bacterial meningitis using simple clinical and laboratory parameters. Youssef FG, Afifi SA, Azab AM, Wasfy MM, Abdel-Aziz KM, Parker M, et al. 2006;55(4), Diagnostic Microbiology and Infectious Disease, pp. 275-278.

[37] 37. Clinical and public health aspects of tuberculous meningitis in children. Doerr CA, Starke JR, Ong LT. 1995;127, Journal of Paediatrics, pp. 27-33.

[38] 38. Diagnostic criteria for tuberculous meningitis and their validation. Ahuja GK, Mohan KK, Prasad K, Behari M. 1994;75, Tubercle and Lung Disease, pp. 149-152.

[39] Streptomycin treatment of tuberculous meningitis. 1948;1, Lancet, pp. 582-96.

[40] Shunt surgery for poor grade patients with tuberculous meningitis and hydrocephalus: Effect of response to external ventricular drainage and other factors on long term outcome. Mathew JM, Rajshekhar V, Chandy MJ. 1998;65, Journal of Neurology, Neurosurgery & Psychiatry, pp. 115-8

[41] 41. Cerebrospinal fluid drug concentrations and the treatment of tuberculosis. Ellard GA, Humphries MJ, Allen BW. 1993;148(3), The American Review of Respiratory Disease, pp. 650-5.

[42] 42. Effect of steroids on cerebrospinal fluid penetration of anti tuberculous drugs in tuberculous meningitis. Kaojarern S, Supmonchai K, Phuapradit P, Mokkhavesa C, Krittiyanunt S. 1991;49(1), Clinical Pharamacology & Therapeutics, pp. 6-12..

[43] 43. Role of individual drugs in the chemotherapy of tuberculous meningitis. DA, Mitchison. 2000;(4), International Journal of Tuberculosis & Lung Disease, pp. 796-806.

[44] 44. Corticosteroids for managing tuberculosis. Prasad K, Singh MB. 2008;(1), Cochrane Database Systematic Review, p. CD002244.

[45] 45. Indian Academy of Pediatrics. Treatment of childhood tuberculosis: Consensus statement of IAP working group. 1997;34, Indain Paediatrics, pp. 1093-6.

[46] Acute Bacterial meningitis. S Aneja, A Agarwal. 1997(34), Indian Paediatrics, pp. 1097-1109.

[47] 47. Prognostic indicators in pyogenic meningitis. Bhat BV, Verma IC, Puri RK, Srinivasan S, Nalini P. 1987;24, Indian Pediatrics, pp. 977-983.

[48] 48. Antibiotic therapy for bacterial meningitis in developing countries. Kumar P, Verma IC. 1993;71, Bulletin of the World Health Organisation, pp. 183-188.

[49] 49. Cellular mechanisms of microbial proteins contributing to invasion of the blood brain barrier. Huang SH, Jong AY. 2001;3, Cellular Microbiology, pp. 277-87.

[50] 50. Tumor necrosis factor, alpha cachectin and interleukin 1 beta initiate meningeal inflammation. Ramilo O, Saez-Llorens X, Mertsola J, et al. 1990;172, The Journal of Experimental Medicine, pp. 497-507.

[51] 51. Pathogenesis of bacterial meningitis. Leib SL, Tauber MG. 1999;13, Infectious Disease Clinics of North America, pp. 527-48.

[52] 52. Reprogaming the host response in bacterial meningitis: how best to improve outcome? van der Flier M, Geelen SP, Kimpen JL, Hoepelman IM, Tuomanen EI. 2003;16, Clinical Microbiology Reviews, pp. 415-29.

[53] 53. Current concepts in the pathogenesis of meningitis caused by streptococcus pneumoniae. Meli DN, Christen S, Leib SL, Tauber MG. 2002;15, Current Opinion in Infectious Diseaes , pp. 253-7.

[54] 54. Dual phase of apoptosis in pneumococcal meningitis. Mitchell L, Smith SH, Braun SH, Herzog KH, Tuomanen EI. 2004;190, The Journal of Infectious Diseases , pp. 2039-46.

[55] 55. Diagnosis and treatment of bacterial meningitis. El Bashir H, Laundy M, Booy R. 2003;88, Archives of Disease in childhood, pp. 6151-20.

[56] 56. Clinical features and prognostic factors in adults with bacterial meningitis. van de Beek D, de Gans J, Spanjaard L, Weisfelt M, Reitsma JB, Vermeulen M. 2004;351, New England Journal of Medicine, pp. 1849-59.

[57] 57. Clinical presentations, diagnosis and prognostic factors of bacterial meningitis . SL, Kaplan. 1999;13, Infectious Disease Clinics of North America, pp. 579-94.

[58] 58. XM, Saez-Lorens. Acute bacterial meningitis beyond the neonata period. Principles and practice of pediatric infectious diseases. Philadelphia : Churchill Livingstone, 2003. p264-71.

[59] 59. Report of the Task Force on Diagnosis and Management of Meningitis. Klein JO, Feigin RD, Me Cracken GH Jr. 1986;78(Suppl), Pediatrics, pp. 959-982.

[60] 60. Lumbar puncture in pediatric bacterial meningitis: drfining the time interval for recovery of cerebrospinal fluid pathogens after parenteral antibioyic pretreatment. Kanegaye JT, Soliemanzadeh P, Bradley JS. 2001;108, Pediatrics, pp. 1169-74.

[61] 61. Bacterial meningits in children. Saez-Llorens X, McCracken Jr, GH. 2003;361, Lancet, pp. 2139-48.

[62] 62. Neurosurgical methods in the treatment of tuberculous meningitis. H, Cairns. 1951;26, Archives of Diseases in Childhood, pp. 376-83.

[63] 63. Ventriculoatrial shunt for hydrocephalus complicating tuberculous meningitis. Murray HW, Bandsetter RD, Levyne MH. 1981;70, American journal of Medicine, pp. 895-8.

[64] 64. Timing of shunt surgery in childhood tuberculous meningitis with hydrocephalus. Kemaloglu S, Ozkan U, Bukte Y, Ceviz A, Ozates M. 2002;37, Paediatric Neurosurgery, pp. 194-8.

[65] 65. Endoscopy for tuberculous hydrocephalus. Figaji AA, Fieggen AG, Peter JC. 2007;23, Child's Nervous System, pp. 79-84.

[66] 66. Endoscopic third ventriculostomy in prepontine-suprasellar tuberculoma with tuberculous meningits hydrocephalus: a case report. Jha D, Khatri P, Choudhary, Sethi R, Kumar S. 2007;43, Paediatric Neurosurgery, pp. 42-6.

[67] 67. Factors affecting the outcome of neuroendoscopy in patients with tuberculous meningitis hydrocephalus: a preliminary study. Jha DK, Mishra V, Choudhary A, Khatri P, Tiwari R, Sural A, et al. 2007;68(1), Surgical Neurology, pp. 35-41. discussion 41-2.

[68] 68. Head injuries in children under 36 months of age: Demography and outcome. Hahn Y S, Chyung C, Barthel M J, et al. 1988;4, Child's Nervous System, pp. 34-40.

[69] Practice guidelines for the management of bacterial meningits . Tunkel AR, Hartman BJ, Kapaln SL, et al. 2004;39, Clinical Infectious Diseases, pp. 1267-84.

[70] Prognostic factors of Tuberculous Meningitis in Adults: A 6 -year retrospective study at a tertiary hospital in north taiwan. Po-Chang Hsu, Chien Chang Yang, Jung-Jr Ye, Po-Yen Huang, Ping-Cherng Chiang, Ming-Hsun Lee. 2010;43(2), Journal of Microbiology, Immunology and Infection, pp. 111-118.

[71] Predictors of long term neurological sequelae of tuberculous menongitis : a multivariate analysis. J kalita, UK Misra, P Ranjan. 2007;14(1), European journal of neurology, pp. 33-37..

[72] Prognostic clinical variables in childhood tuberculous meningitis: an experience from mumbai, india. Sunil Karande, Vishal gupta, Madhur Kulkarni, Anagha Joshi. 2005;53(2), pediatrics, pp. 191-196.

[73] Outcome of ventriculoperitoneal shunt placement in Grade IV tubercular meninigits with hydrocephalus : a retrospective analysisi in 95 patients. Srikantha U, Morab JV, Saatry S, Abraham R. Balasubramanian A, Somanna S, Devi I, Pandey P. 441-460, Bangalore : Journal of Neurosurgery Paediatrics , 2009, Vol. Aug 4(2).

[74] Misra UK, Kalita J, Srivatsava M, Mandal SK. Prognosis of tuberculous meningitis: a multivariate analysis. Journal of Neurological Sciences . 1996; 137: 57- 61

[75] PaganiniH, Gonzales F, Santandar C, Casimir, L, Berberian G, Rosanova M T. Tubercolous meningitis in children : clinical features and outcome in 40 cases. Scan J Infec Dis 2000; 32: 41-45

[76] Palur R, Rajsekhar V, Chandy MJ, Joseph T, Abraham J. Shunt Surgery for hydrocephalous in Tuberculous meningitis. A long term follow up study. J Neurosurgery 1991;74:64-9

[77] Singh D, Kumar S. Ventriculopertineal Shunt in Post Tubercular Hydrocephalous. Paedtr. Neurosurg 2002;37:194-8

[78] Sil K, Chaterjee S. Shunting in Tuberculous meningitis : A neurosurgeon's nightmare. Child Nerv Syst 2008;24:1029-32

[79] Rajshekhar V. Management of hydrocephalus in patients with tuberculous meningitis. Neurol India. 2009 Jul-Aug;57(4):368-74. doi: 10.4103/0028-3886.55572.

[80] DPE Kingsley, WA Hendrickse, B E Kendell, M Swash, V Singh. Tuberculous Meningitis : role of CT in management and prognosis Journal of Neurology, Neurosurgery and Psychiatry 1987;50:30-36

[81] Smith HV. Tuberculous meningistis. Int J Neurol 1964;4:134-57

[82] Delage G, Dusseault M. Tuberculous meningitis in children; a retrospective study of 79 patients with an analysis of prognostic factors. Can Med Assoc J 1979;120:305-9

[83] Bateman DE, Newman PK, Foster JB. A retrospective survery of proven cases of tuberculous meningitis in the Northern Region 1970-80J R Coll Phys Lond 1983;17:106-10

[84] Tandon PN, Tandon HD. Tuberculous meningistis. A continuing challenge. J All India Inst Med Sci 1975;2:99-104

[85] Verdon R, Chevret S, Laissy J-P, Wolff M. Tuberculous meningistis in adults: Review of 48 cases, Clin Infect Dis 1996;22:982-988

[86] S Ambedkar, S.Dwarakanath, B.A.Chandramouli, S.Sampath, B.Indira Devi and P.Pandey. Does CSF Composition predict shunt malfunction in Tuberculous Meningitis. Indian Journal of Tuberculosis 2011;58:77-81

APPENDIX I - Ethical Committee Approval

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To

Dr.K.Balasubramani,
Post Graduate in Neurosurgery,
Madras Medical College,Chennai -3

Dear Dr.K.Balasubramani,

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "Factors predicting outcome after surgery in post meningitic Hydrocephalus" No.23022013.

The following members of Ethics Committee were present in the meeting held on 05.02.2013 conducted at Madras Medical College, Chennai -3.

1. Dr.SivaKumar, MS FICS FAIS	--- Chairperson
2. Prof. R. Nandhini MD Director, Instt. of Pharmacology ,MMC, Ch-3	-- Member Secretary
3. Prof. Shyamraj MD Director i/c , Instt. of Biochemistry , MMC, Ch-3	-- Member
4. Prof. P. Karkuzhali. MD Prof., Instt. of Pathology, MMC, Ch-3	-- Member
5. Prof. A. Radhakrishnan MD Prof of Internal Medicine, MMC, Ch-3	-- Member
6. Prof. S. Deivanayagam MS Prof of Surgery, MMC, Ch-3	-- Member
7. Thiru. S. Govindsamy. BABL	-- Lawyer
8. Tmt. Arnold Soulina MA MSW	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

R Nandini 20/3/13
Member Secretary, Ethics Committee

APPENDIX II - Copy of Informed Consent ஆராய்ச்சி ஒப்புதல் கடிதம்

ஆராய்ச்சி ஒப்புதல் கடிதம்

ஆராய்ச்சி தலைப்பு: மூளைக் காய்ச்சலால் மூளையில் நீர்கோர்திருத்தலுக்கான (POST MENINGITIC HYDROCEPHALUS) அறுவை சிகிச்சையின் பின் முடிவுகளை பாதிக்கும் காரணிகள் பற்றிய ஆய்வு.

பெயர்: வயது: பால்:

ஆராய்ச்சி சேர்க்கை எண்:

- ராஜீவ்காந்தி அரசு பொது மருத்துவக்கல்லூரி மற்றும் அரசு பொது மருத்துவமனையின் நரம்பியல் அறுவை சிகிச்சைத் துறையில் "மூளைக் காய்ச்சலால் மூளையில் நீர்கோர்திருத்தலுக்கான (POST MENINGITIC HYDROCEPHALUS) அறுவை சிகிச்சையின் பின் முடிவுகளை பாதிக்கும் காரணிகள்" பற்றிய ஆய்வு நடைபெறுகிறது என்பதை அறிந்துகொண்டேன்.
- சி.டி.ஸ்கேன் மற்றும் எம்.ஆர்.ஐ. ஸ்கேன் ஆகியவற்றின் அடிப்படையில் இந்த ஆய்வு நடைபெறுகிறது என்பதையும் மேலும் அறுவை சிகிச்சையின்போது நேரடியாக பார்க்கப்படுவதை வைத்தும் ஆய்வு நடைபெறுகிறது என்பதையும் அறிந்துகொண்டேன்.
- இவ்வாய்வில் கலந்துகொள்பவர்களின் சொந்த தகவல்கள் ரகசியமாக பாதுகாக்கப்படும் என்பதையும் இந்த ஆய்வின் முடிவுகளை பிரசுரிக்கும்போது அல்லது வெளியிடும்போதோ தங்களின் தகவல்கள் ஏதும் வெளியிடப்படாது என்பதையும் அறிந்துகொண்டேன்.
- இந்த ஆராய்ச்சியிலிருந்து எந்த நேரமும் பின்வாங்கலாம் என்றும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் அறிந்துகொண்டேன்.
- இந்த ஆய்வில் பங்குபெற அல்லது விலக்கிக்கொள்ள எனக்கு முழு சுதந்திரம் உண்டு என்பதையும், இந்த ஆய்வில் இருந்து நான் விலகிக்கொண்டாலும் எனக்கு கிடைக்கவேண்டிய சிகிச்சை தொடர்ந்து கிடைக்கும் என்பதையும் அறிந்துகொண்டேன்.
- இந்த ஆராய்ச்சியின் விவரங்களும், அதன் நோக்கங்களும் எனக்கு தெளிவாக விளக்கப்பட்டது. எனக்கு விளக்கப்பட்ட விவரங்களை புரிந்துகொண்டு இந்த ஆய்வில் கலந்துகொள்ள சம்மதிக்கிறேன்.
- இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின்பேரில்தான் பங்குபெறுகிறேன்.

கையொப்பம்

APPENDIX III - Copy of Patient Information Sheet

ஆராய்ச்சித் தகவல் தாள்

ஆராய்ச்சி தலைப்பு

மூளைக் காய்ச்சலால் மூளையில் நீர்கோர்திருத்தலுக்கான (POST MENINGITIC HYDROCEPHALUS) அறுவை சிகிச்சையின் பின் முடிவுகளை பாதிக்கும் காரணிகள்

பெயர் : வயது :
ஆராய்ச்சி சேர்க்கை எண் : தேதி :

இந்த ஆராய்ச்சியின் விவரங்களைக் கொண்ட தகவல் தாளைப் பெற்றுக்கொண்டேன்.

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கமும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்துகொண்டு எனது சம்மதத்தை தெரிவிக்கிறேன்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின்பேரில்தான் பங்கு பெறுகின்றேன். இந்த ஆராய்ச்சியில் இருந்து நான் எந்நேரமும் பின் வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்துகொண்டேன்.

நான் என்னுடைய சுய நினைவுடனும் மற்றும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக்கொள்ள சம்மதிக்கிறேன்.

ஆராய்ச்சியாளர் மற்றும் அவரைச் சார்ந்தவர்களோ, நெறிமுறைக்குழு உறுப்பினர்களோ நான் இந்த ஆராய்ச்சியில் இருந்து விலகினாலும் என்னுடைய அனுமதியின்றி எனது உடல்நிலை குறித்த தகவல்களை இந்த ஆராய்ச்சிக்கோ இது தொடர்பான வேறு ஆராய்ச்சிகளுக்கோ பயன்படுத்திக்கொள்ள முடியும் என்று புரிந்து கொண்டு சம்மதம் அளிக்கிறேன். ஆனாலும் என்னுடைய அடையாளம் வெளியிடப்படமாட்டாது என்று புரிந்துகொள்கிறேன்.

இந்த ஆராய்ச்சியின் தகவல்களையும் முடிவுகளையும் அறிவியல் நோக்கத்திற்காக பயன்படுத்துவதற்கு நான் அனுமதிக்கிறேன். நான் ஆராய்ச்சியில் பங்குபெற சம்மதிக்கிறேன்.

பங்கேற்பவரின் பெயர் **பங்கேற்பவரின் கையொப்பம் (அல்லது) கட்டைவிரல் ரேகை**

ஆய்வாளர் பெயர் **ஆய்வாளரின் கையொப்பம்**

இடம் தேதி

APPENDIX IV - COPY OF PROFORMA USED

INSTITUTE OF NEUROLOGY
GOVERNMENT GENERAL HOSPITAL, CHENNAI
PROFORMA

PROFORMA

FACTORS PREDICTINGOUTCOME AFTER SURGERY IN POST MENINGITIC HYDROCEPHALUS

NAME: AGE: IP NO:

ADDRESS: DOA:

DOD:

DOS:

COMPLAINTS :

HISTORY:

EXAMINATION:

CT SCAN BRAIN:

CSF STUDY:

SURGERY:

POST OP PERIOD:
Post OP CT Brain:
COD:
FOLLOW UP AFTER 12 WEEKS:

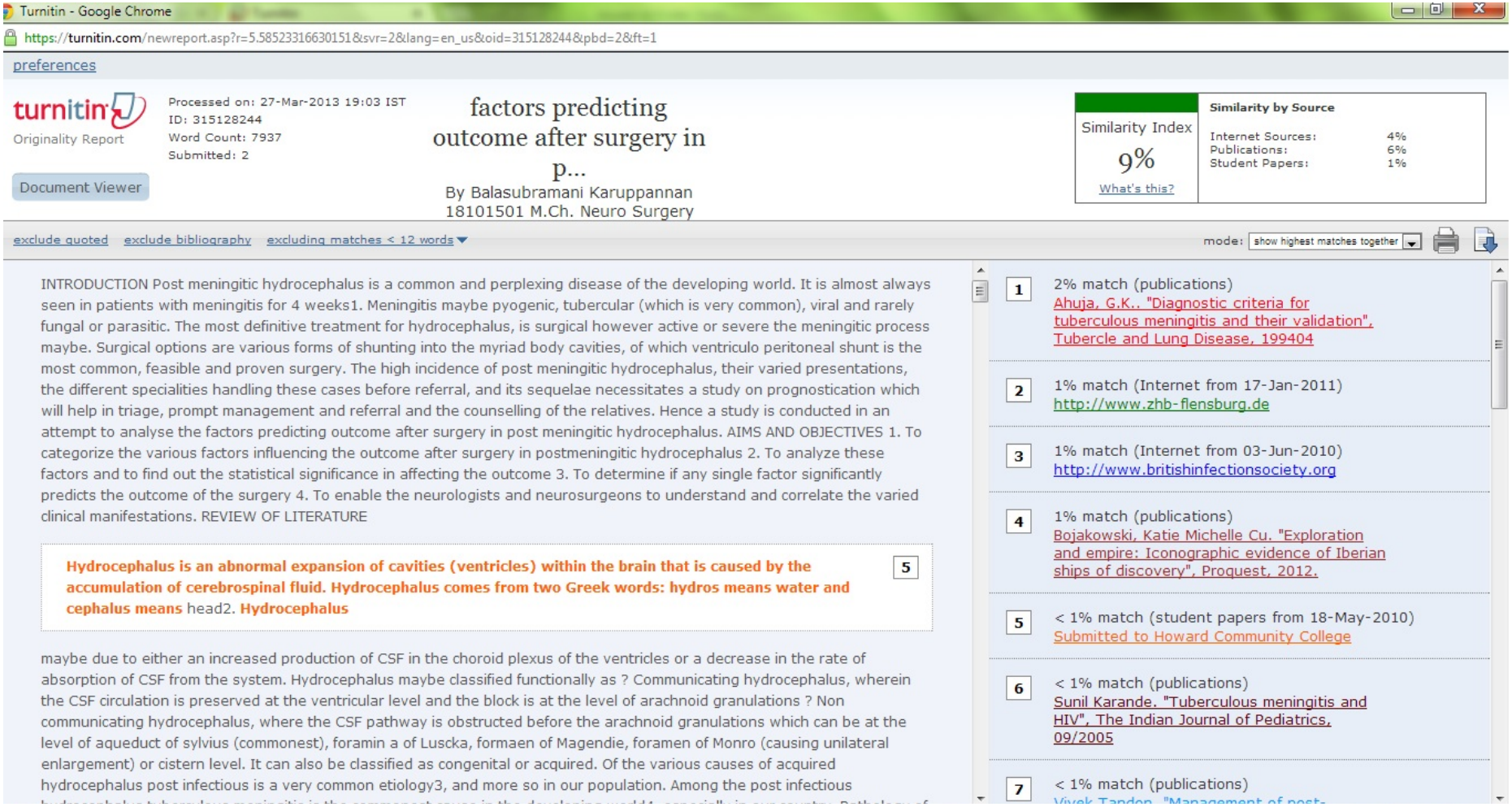
APPENDIX V –MASTER CHART

S.No	Age in years	Sex	Duration of Fever	GCS on Admission	Seizures	Focal Deficits	Exudates in CT Scan	Infarcts in CT Scan	Tuberculoma in CT Scan	Time Interval for Surgery	CSF Lymphocytosis	CSF Protein	CSF Protein 200	CSF Sugar	CSF Gram Stain	CSF Aerobic C/S	CSF Anerobic C/S	CSF AFB stain	CSF AFB C/S	CSF Fungal Stain	Vellore stage	Outcome
1	0.58	m	25	15	No	No	Yes	Yes	No	<48	Yes	60	<200	55	No	No	No	No	No	No	1	Recovery
2	0.17	f	18	8	Yes	Yes	Yes	No	No	>48	Yes	426	>200	30	No	No	No	No	No	No	4	Death
3	1	m	29	15	No	No	Yes	No	No	<48	Yes	161	<200	44	No	No	No	No	No	No	1	Recovery
4	0.17	f	24	15	No	Yes	Yes	Yes	No	<48	Yes	107	<200	38	No	No	No	No	No	No	2	Death
5	0.42	m	26	9	Yes	No	Yes	No	Yes	>48	Yes	232	>200	52	No	No	No	Yes	No	No	3	Death
6	0.17	m	19	15	Yes	No	Yes	No	Yes	<48	Yes	107	<200	58	No	No	No	No	No	No	1	Death
7	1	m	14	9	Yes	Yes	Yes	No	No	>48	Yes	365	>200	41	No	No	No	No	No	No	3	Sequelae
8	0.17	f	29	15	No	Yes	No	No	No	<48	Yes	559	>200	47	No	No	No	No	No	No	2	Sequelae
9	1	m	25	9	Yes	No	No	Yes	No	<48	Yes	156	<200	54	No	No	No	Yes	No	No	3	Recovery
10	0.67	f	27	15	Yes	Yes	No	No	No	>48	Yes	191	<200	48	No	No	No	No	No	No	2	Death
11	2	m	17	8	Yes	Yes	No	No	No	>48	Yes	127	<200	39	No	No	No	No	No	No	4	Sequelae
12	0.92	f	22	15	No	No	Yes	No	No	<48	Yes	283	>200	30	No	No	No	No	No	No	1	Recovery
13	0.58	f	25	9	Yes	No	Yes	No	No	>48	Yes	264	>200	44	No	No	No	Yes	No	No	3	Death
14	0.17	f	27	15	Yes	Yes	Yes	No	No	<48	Yes	418	>200	44	No	No	No	No	No	No	2	Sequelae
15	5	m	24	8	No	No	Yes	No	No	<48	Yes	394	>200	31	No	No	No	No	No	No	4	Recovery
16	0.17	f	28	9	Yes	No	Yes	No	No	<48	Yes	466	>200	57	No	No	No	No	No	No	3	Death
17	0.33	m	20	15	Yes	No	No	Yes	No	>48	Yes	269	>200	38	No	No	No	No	No	No	1	Sequelae
18	1	m	23	8	Yes	Yes	No	No	No	>48	Yes	340	>200	31	No	No	No	No	No	No	4	Death
19	5	m	17	15	Yes	No	Yes	No	Yes	<48	Yes	187	<200	58	No	No	No	Yes	No	No	1	Recovery
20	0.33	f	22	15	No	Yes	Yes	No	No	>48	Yes	276	>200	59	No	No	No	No	No	No	2	Death
21	6	f	22	15	Yes	Yes	Yes	No	No	<48	Yes	243	>200	49	No	No	No	No	No	No	2	Recovery

S.No	Age in years	Sex	Duration of Fever	GCS on Admission	Seizures	Focal Deficits	Exudates in CT Scan	Infarcts in CT Scan	Tuberculoma in CT Scan	Time Interval for Surgery	CSF Lymphocytosis	CSF Protein	CSF Protein 200	CSF Sugar	CSF Gram Stain	CSF Aerobic C/S	CSF Anerobic C/S	CSF AFB stain	CSF AFB C/S	CSF Fungal Stain	Vellore stage	Outcome
22	0.42	f	16	8	Yes	No	No	Yes	No	<48	Yes	267	>200	43	No	No	No	Yes	No	No	4	Death
23	0.58	m	26	15	No	No	No	No	Yes	<48	Yes	479	>200	43	No	No	No	No	No	No	1	Recovery
24	0.33	f	24	8	No	Yes	Yes	No	Yes	>48	Yes	483	>200	43	No	No	No	No	No	No	4	Death
25	2	m	22	15	No	No	Yes	No	No	<48	Yes	79	<200	32	No	No	No	No	No	No	1	Sequelae
26	0.17	f	22	15	No	Yes	No	Yes	No	<48	Yes	397	>200	30	No	No	No	No	No	No	2	Sequelae
27	0.25	f	28	9	No	No	Yes	No	No	>48	Yes	369	>200	32	No	No	No	No	Yes	No	3	Death
28	0.17	f	19	15	No	No	Yes	Yes	No	<48	Yes	74	<200	43	No	No	No	No	No	No	1	Recovery
29	2	m	22	8	No	Yes	Yes	No	No	>48	Yes	390	>200	32	No	No	No	No	No	No	4	Sequelae
30	11	m	29	9	No	Yes	Yes	No	No	>48	Yes	496	>200	35	No	No	No	No	No	No	3	Sequelae
31	0.42	m	26	15	No	No	Yes	No	No	<48	Yes	368	>200	42	No	No	No	No	No	No	1	Recovery
32	4	f	24	15	No	Yes	Yes	No	No	<48	Yes	139	<200	32	No	No	No	No	No	No	2	Recovery
33	14	m	23	9	No	No	No	No	No	<48	Yes	140	<200	35	No	No	No	No	No	No	3	Recovery
34	12	m	29	15	No	No	Yes	No	No	>48	Yes	416	>200	36	No	No	No	No	No	No	1	Recovery
35	3.5	f	29	9	No	No	No	Yes	No	>48	Yes	558	>200	58	No	No	No	No	No	No	3	Recovery
36	0.25	f	29	15	No	No	Yes	No	No	<48	Yes	129	<200	34	No	No	No	No	No	No	1	Recovery
37	0.17	m	23	9	No	Yes	No	Yes	Yes	<48	Yes	240	>200	42	No	No	No	No	No	No	3	Sequelae
38	0.92	m	21	15	No	No	No	No	No	<48	Yes	182	<200	49	No	No	No	No	No	No	1	Recovery
39	0.42	f	18	15	No	Yes	Yes	No	No	<48	Yes	198	<200	53	No	No	No	No	No	No	2	Recovery
40	3.5	f	25	15	No	Yes	No	No	No	>48	Yes	157	<200	46	No	No	No	No	No	No	2	Recovery
41	0.25	m	17	8	Yes	No	Yes	No	No	<48	Yes	72	<200	35	No	No	No	Yes	No	No	4	Death
42	1.5	m	16	15	No	Yes	Yes	No	No	<48	Yes	399	>200	46	No	No	No	No	No	No	2	Recovery
43	5	f	26	15	No	No	No	No	No	<48	Yes	354	>200	43	No	No	No	No	No	No	1	Recovery
44	2	f	28	9	No	No	No	No	No	<48	Yes	138	<200	36	No	No	No	No	No	No	3	Recovery
45	0.25	f	26	15	No	No	Yes	No	No	<48	Yes	531	>200	50	No	No	No	No	No	No	1	Recovery
46	6	m	24	15	No	Yes	Yes	No	No	<48	Yes	306	>200	31	No	No	No	Yes	No	No	2	Sequelae

S.No	Age in years	Sex	Duration of Fever	GCS on Admission	Seizures	Focal Deficits	Exudates in CT Scan	Infarcts in CT Scan	Tuberculoma in CT Scan	Time Interval for Surgery	CSF Lymphocytosis	CSF Protein	CSF Protein 200	CSF Sugar	CSF Gram Stain	CSF Aerobic C/S	CSF Anerobic C/S	CSF AFB stain	CSF AFB C/S	CSF Fungal Stain	Vellore stage	Outcome
47	0.58	m	17	8	Yes	Yes	Yes	No	No	>48	Yes	165	<200	42	No	No	No	No	No	No	4	Death
48	9	m	14	9	No	No	Yes	No	No	<48	Yes	184	<200	40	No	No	No	No	No	No	3	Recovery
49	0.58	f	16	8	Yes	Yes	Yes	No	No	>48	Yes	530	>200	48	No	No	No	No	No	No	4	Death
50	0.92	f	17	9	No	Yes	Yes	Yes	No	<48	Yes	125	<200	35	No	No	No	No	No	No	3	Sequelae
51	0.58	f	28	9	No	No	Yes	No	No	<48	Yes	175	<200	43	No	No	No	No	No	No	3	Recovery
52	0.25	f	22	8	Yes	No	Yes	No	Yes	>48	Yes	277	>200	54	No	No	No	Yes	No	No	4	Death
53	2	f	25	15	No	No	Yes	No	No	<48	Yes	195	<200	40	No	No	No	No	No	No	1	Recovery
54	0.42	m	21	15	No	Yes	Yes	No	No	>48	Yes	173	<200	31	No	No	No	No	No	No	2	Recovery
55	7	f	15	9	No	Yes	Yes	No	No	<48	Yes	297	>200	53	No	No	No	No	No	No	3	Sequelae
56	6	m	18	15	No	No	Yes	No	No	<48	Yes	544	>200	30	No	No	No	No	No	No	1	Recovery
57	0.17	m	17	8	Yes	Yes	No	No	No	<48	No	920	>200	12	Yes	No	No	No	No	No	4	Death
58	5	f	19	9	Yes	Yes	No	No	No	>48	No	740	>200	30	Yes	Yes	No	No	No	No	3	Death
59	1.5	m	17	15	No	No	No	No	No	<48	No	480	>200	20	Yes	Yes	No	No	No	No	1	Recovery
60	7	m	9	15	No	No	No	No	No	<48	No	150	<200	50	Yes	No	No	No	No	No	1	Recovery
61	0.5	f	8	15	No	No	No	No	No	>48	No	300	>200	60	Yes	Yes	No	No	No	No	1	Recovery
62	4.5	m	18	15	No	Yes	Yes	No	No	<48	No	410	>200	24	Yes	No	No	No	No	No	2	Sequelae
63	0.33	f	8	9	Yes	No	No	No	No	<48	No	250	>200	22	Yes	Yes	No	No	No	No	3	Sequelae
64	3	f	7	9	No	No	Yes	No	No	<48	No	300	>200	36	Yes	Yes	No	No	No	No	3	Recovery
65	6	m	18	8	No	No	No	Yes	No	<48	No	450	>200	38	Yes	No	No	No	No	No	4	Recovery

APPENDIX VI – TURNITIN REPORT FOR PLAGIARISM



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INTRODUCTION Post meningitic hydrocephalus is a common and perplexing disease of the developing world. It is almost always seen in patients with meningitis for 4 weeks1. Meningitis maybe pyogenic, tubercular (which is very common), viral and rarely fungal or parasitic. The most definitive treatment for hydrocephalus, is surgical however active or severe the meningitic process maybe. Surgical options are various forms of shunting into the myriad body cavities, of which ventriculo peritoneal shunt is the most common, feasible and proven surgery. The high incidence of post meningitic hydrocephalus, their varied presentations, the different specialities handling these cases before referral, and its sequelae necessitates a study on prognostication which will help in triage, prompt management and referral and the counselling of the relatives. Hence a study is conducted in an attempt to analyse the factors predicting outcome after surgery in post meningitic hydrocephalus. AIMS AND OBJECTIVES 1. To categorize the various factors influencing the outcome after surgery in postmeningitic hydrocephalus 2. To analyze these factors and to find out the statistical significance in affecting the outcome 3. To determine if any single factor significantly predicts the outcome of the surgery 4. To enable the neurologists and neurosurgeons to understand and correlate the varied clinical manifestations. REVIEW OF LITERATURE

Hydrocephalus is an abnormal expansion of cavities (ventricles) within the brain that is caused by the accumulation of cerebrospinal fluid. Hydrocephalus comes from two Greek words: hydros means water and cephalus means head2. **Hydrocephalus** 5

maybe due to either an increased production of CSF in the choroid plexus of the ventricles or a decrease in the rate of absorption of CSF from the system. Hydrocephalus maybe classified functionally as ? Communicating hydrocephalus, wherein the CSF circulation is preserved at the ventricular level and the block is at the level of arachnoid granulations ? Non communicating hydrocephalus, where the CSF pathway is obstructed before the arachnoid granulations which can be at the level of aqueduct of sylvius (commonest), foramin a of Luscka, formaen of Magendie, foramen of Monro (causing unilateral enlargement) or cistern level. It can also be classified as congenital or acquired. Of the various causes of acquired hydrocephalus post infectious is a very common etiology3, and more so in our population. Among the post infectious

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